"I would have everie man write what he knowes and no more."—MONTAIGNE

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EDITORIAL

TO REVIEW or not to review, to sign or not to sign? This is an old question, but it has recently cropped up in the correspondence columns of the *British Medical Journal*, where it is very evident that "Doctors disagree" and doctors of great experience, at least in so far as the signing or not signing. Who then shall decide?

If we take John Snow's advice this question of signing would not arise. In Sir Benjamin Ward Richardson's essay on John Snow published in the *Asclepiad* of 1866 and reprinted in the *British Journal of Anaesthesia* of October 1952, he has this to say: "He thought severely of the reviewer's art, and would never of late review any book critically. If a book were good, it carried the review of its own merits. It it were bad, it were better left untouched." However, it is now the custom to review books and it is doubtful if authors would have it otherwise; to be ignored is *usually* worse than to be criticized. We decided some years ago to sign our reviews and in that we have the support of Dr. Lillingston who in his letter to the *B.M.J.*, August 20, 1955, quotes Pascal as saying "*Diseur de bons mots, mauvais caractère*". The obvious *double entendre* suggests that the French reviewer who is witty at the expense of his author is a bad man, but in England it is the sycophant who merits that name. How hard then is the lot of the reviewer, how difficult his task. Between the devil of an author and the deep sea of his readers resentment lies on either hand. The author's is easily aroused and the reader who has bought a book on the reviewer's recommendation and finds it not to his taste is very naturally annoyed. By the time a man writes a book on a technical subject he is probably somewhat acquainted with it and has overcome that degree of modesty with which nature originally endowed him sufficiently to publish it. A glance at the preface will frequently reveal his reason for so doing, but so mixed are human motives that the reviewer who based his criticism on this alone, however favourable, would be likely to find that the brevity of his remarks had disappointed the author and failed to give his readers the information they desired.

Two reviews of a book by a well-known author read as follows:

(1) "This ripe and delightful book is in the author's most brilliant mood."

(2) "The best thing about this otherwise disappointing little book is the sincerity that keeps peeping through of Mr. X's delight in the homeland of his ancestors."

Could one hesitate?

Yet it has not been an uncommon experience of late to buy books favourably reviewed and find that we have spent our money on bread not even fit to be cast upon the waters. Perhaps some of our

readers would care to air their views on this subject?

FOR a full account of Dr. H. E. Boyle, whose portrait appears on the front cover of this issue, our readers are referred to the article by his friend Dr. Hadfield which appeared in this journal in our April 1950 number. Here we may refer to his dates. He was born in 1875, qualified in 1901, spent his professional life as anaesthetist at Bart's; he retired in 1940 and died the following year. Every anaesthetist knows Boyle's apparatus and the Davis Boyle gag. Dr. Boyle was one of the pioneers of the new mechanical age when the old highly skilled methods of anaesthesia gave place to the use of machines and the consequent easing of the burden of the anaesthetist, owing to the increased safety of the patient which the machines brought about.

BOOK REVIEW

Saddle Block Anaesthesia. By Ray T. Parmley, M.D. Published by Blackwell Scientific Publications. Price 18s.

This is another of the Thomas series, therefore delightful to handle and a joy to possess. The contents appear to be a collection of the knowledge and wisdom acquired in the last forty years or so in the very limited field with which the book deals; that is to say it is a good book, it is sound, the advice given can be relied on, it is handy and compact. The doctor on his own, cut off from professional assistance, with little knowledge or experience of spinal anaesthesia in obstetrics, would find it an invaluable guide, and a quick method of obtaining a sound grasp of "saddle block" anaesthesia—its indications and limitations. Perhaps this is enough to justify its publication.

"Is your journey really necessary?" is a question we were often asked during the war, and it recurs to one's mind in connection with this book, for the simple reason that I can find nothing new in it. Neither the giving of a small concentrated heavy solution in the sitting position nor the mixing of a vasoconstrictor in the shape of epinephrine with the anaesthetic is new. Both indeed are more than twenty years old. Dr. Parmley seems to be aware of all this but claims not for himself but for Dr. Adriani the invention of the term "saddle block". Well, Well!

There are one or two mistakes. Piperocaine appears as pipeorcaine on page 23. The legend of figure 5 states that the dural sac ends at L2. Is this a unique phenomenon or should it be S2, as custom expects and the figure itself indicates?

Whatever may be thought of the necessity or desirability of publishing a book on this small field of spinal anaesthesia it gives Dr. Adriani in the Preface a chance to express his sound common sense on the subject. He claims to be neutral, a real scientific observer of facts, neither therefore an ardent advocate nor a violent opponent of spinal anaesthesia. This is what he says:

> The tendency to abandon the block is due to the present day trend to avoid spinal anaesthesia because of medico-legal considerations rather than deficiency of the method. The present day attitude of the public as a whole appears to be one of attempting to get something for nothing. Certain insurance companies, underwriting liability coverage for physicians, except coverage for accidents due to spinal anaesthesia in their policies. Thus a physician fearing the consequences of this dictum no longer unequivocally prescribes what he thinks best for the patient. Instead lay groups, who obviously are less versed in the subject, are inadvertently and no doubt unintentionally, meddling and formulating policy concerning selection of anaesthesia. Physicians thus have been forced to abandon a method of anaesthesia which might be more advantageous than others to the patient.

That surely is a fair statement of the case. The situation in this country is much the same. Is it satisfactory? Is it sane? Reliable statistics of spinal anaesthesia can show—have shown—even better results than those shown by orthodox methods of general anaesthesia current in the last twenty years. Is the better method to be abandoned because its failures are dramatically obvious, whilst those of the other methods are mercifully hidden? *E. Falkner Hill*

Brit. J. Anaesth. (1955), **27**, 471

THE EFFECT OF ANAESTHETICS ON THE RESPONSE TO STIMULATION OF THE ULNAR NERVE IN MAN

BY

R. CECIL BROWN

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GENERAL anaesthetic drugs are believed to have little effect on the peripheral nerves (Hewer, 1953). On the other hand, some anaesthetics, e.g. diethyl ether, are reputed to have a peripheral curare-like action (Gross and Cullen, 1943). It became necessary in the course of other work, to re-examine this position.

It was thought that a suitable test for peripheral blocking action would be provided by observing an alteration in the response of muscles to electrical stimulation of the nerve supplying them. Patients received one of two anaesthetic mixtures; either pethidine / thiopentone / nitrous oxide, or nitrous oxide/ether.

TECHNIQUE

Electrical stimulation was applied by a surface electrode to the ulnar nerve at the elbow. A low-frequency stimulator of conventional design was used, giving one-millisecond impulses at a selected rate. As it was desired to compare the anaesthetized with the unanaesthetized state, the first series of experiments was designed to determine the minimal voltage at which a twitch could be elicited in the supplied muscles using single shocks. With pethidine/thiopentone/nitrous oxide, this involved only two observations, one made with the patient conscious and one after the induction of anaesthesia. With nitrous oxide/ether additional observations were made in each plane of anaesthesia attained. The planes of anaesthesia were judged clinically.

In a second series of experiments the ability of the muscles supplied by the ulnar nerve to maintain a tetanus imposed by applying 16 impulses/second for about 5 seconds was assessed. There was some difficulty in evolving a suitable form of apparatus for demonstrating maintained tetanus. Three types of apparatus were tested.

(1) A large rubber ball lay between the palm of the hand and a modified "cock-up" splint. Contraction of the muscles supplied by the ulnar nerve caused an increase of pressure in the bulb. This pressure could be transmitted to a tambour carrying a lever writing on a kymograph drum. This was soon abandoned because it did not record adequately contraction of only one finger.

(2) Flexion of the little finger was arranged to compress an inflated tambour. The pressure-change was transmitted to a second tambour, which carried a lever writing on a kymograph. The tambours were partially inflated at the outset with a sphygmomanometer bulb (figs. 1, 1A). This apparatus was an improvement on the preceding one. However, it was not easy to show more than a certain range

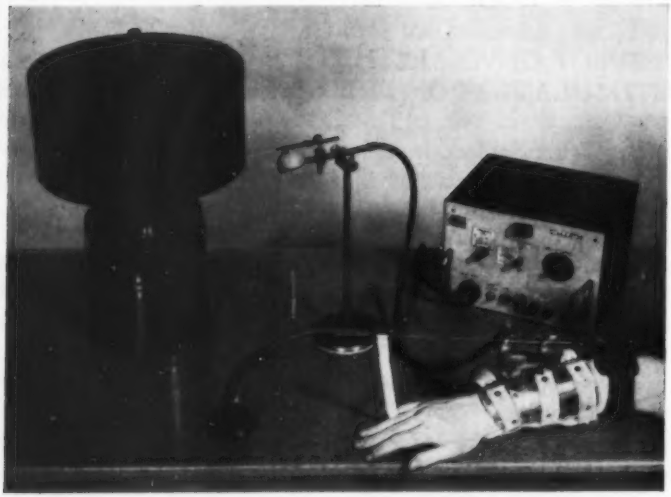

FIG. 1

Experimental set-up showing second form of apparatus. Electrical shocks are conveyed from the stimulator to the ulnar nerve. Contraction of the little finger causes an increase of pressure in the tambour carried on the back of the forearm. This pressure is carried to a second tambour which, by means of a lever, writes on a drum.

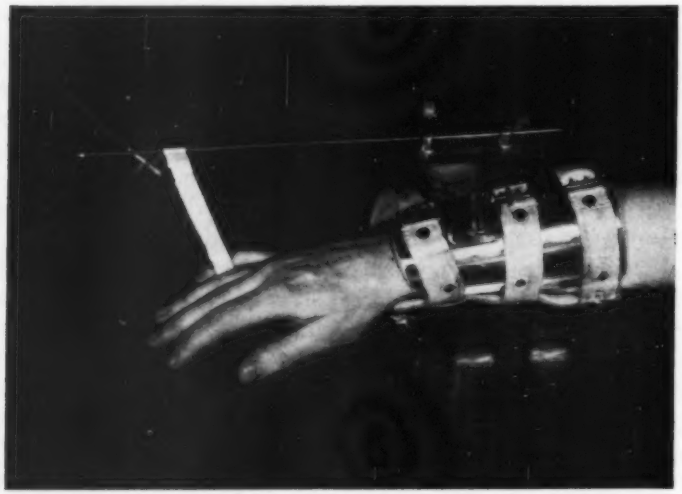

FIG. 1A

Detail from above.

of movement, and there was sometimes a certain asymmetry in the transmission of pressure from one tambour to the other.

(3) With the third apparatus the movement produced by flexion of the little finger was transmitted by a piano-wire running in a flexible metal sheath directly to a lever which produced deflections recorded on a kymograph drum (fig. 2). Movement of the wire was opposed by a cylindrical compression-spring. The apparatus was so constructed that different strengths of compression-spring could be inserted. By arranging the kymograph on the floor, so that the wire was reasonably straight, frictional effects could be reduced until they appeared to be unimportant.

With all these forms of apparatus used in the recording of tetanic contractions, supramaximal stimulation was used. It follows that no observations could be made when the patient was conscious. Observations with the pethidine/thio-

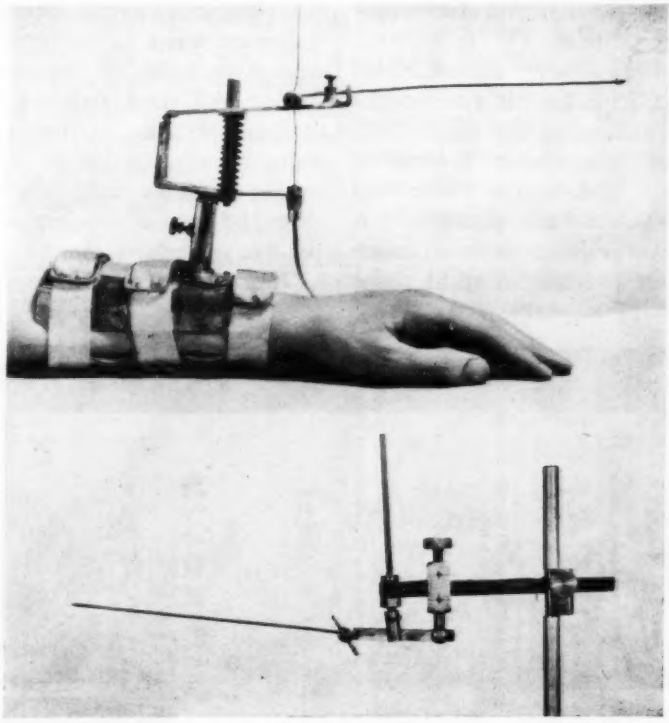

FIG. 2

The third form of apparatus used. The little finger is fastened by adhesive tape to the long lever carried on the fore-arm. Contraction of the finger moves the piano-wire to produce movement of the second lever, which writes on a drum. The movement of the finger is opposed by a cylindrical compression-spring.

pentone/nitrous oxide patients were therefore restricted to showing whether sustained tetanus was possible after induction of anaesthesia. With the nitrous oxide/ether patients, observations could be made in each plane of anaesthesia attained.

RESULTS

First series. Fifteen patients were given pethidine / thiopentone / nitrous oxide. There was no significant alteration in the voltage at which it was possible to elicit a minimal response before and after establishment of anaesthesia.

Twelve patients were anaesthetized with nitrous oxide/ether. It was thought justifiable to proceed to the third plane of third stage anaesthesia in ten of these patients. The fourth plane was attained in five of the patients. A slight rise in threshold to stimulation occurred in five patients in third plane anaesthesia, and a further slight rise took place in two of the patients in fourth plane anaesthesia. Such inconclusive results were highly unsatisfactory. In addition, there was a clinical impression of a decrease in the response to stimulation in the deeper planes of anaesthesia. Though an attempt was always made to obtain the minimal response of the supplied muscles, the response in the deeper planes of anaesthesia was less vigorous than in the earlier planes. The second series of experiments was designed to test the truth of this impression.

Second series. Another fifteen patients were given pethidine/thiopentone/nitrous oxide. All these patients were able to maintain tetanic contraction after the induction of anaesthesia. There was no falling-off in the amplitude of contraction when the burst of impulses was maintained for five seconds or more.

Twenty-two patients were also anaes-

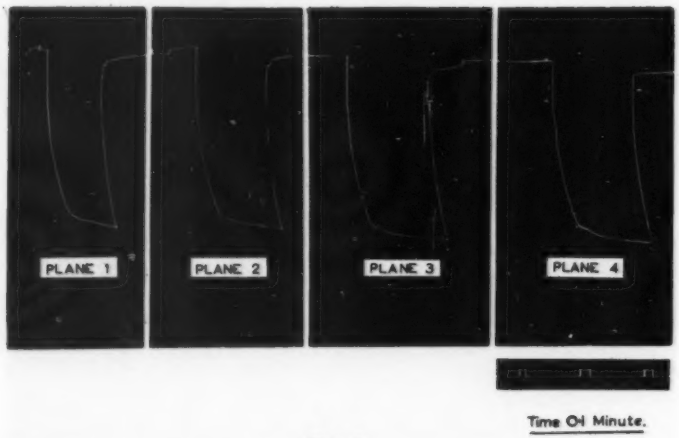

FIG. 3

Male, aged 35. Given nitrous oxide/ether. Third apparatus. Showing the deflections produced by supramaximal stimulation of the ulnar nerve in third stage anaesthesia. (See note in text on failure of some of up-strokes to return immediately to base-line.)

thetized with nitrous oxide/ether. These patients were also able to maintain tetanic contraction during long bursts of stimuli. When using the second form of apparatus it was at first thought that there was some diminution in the force of contraction in the deeper planes of ether anaesthesia—shown by a decreased deflection in these planes. Subsequent experience attributed such an effect to a change in the base-line. The last fourteen patients were all tested with the third form of apparatus, and in no case was there any convincing decrease in the deflection in the deeper planes of ether anaesthesia. Two sample records are shown (figs. 3, 4). In figure 3 it will be noted that a minor instrumental fault had developed, so that in some deflections the up-stroke has not returned immediately to the base-line. This, however, does not affect the validity of the observation that not only is tetanus maintained, but there is no significant decrease in the total deflection produced in the deeper planes of ether anaesthesia.

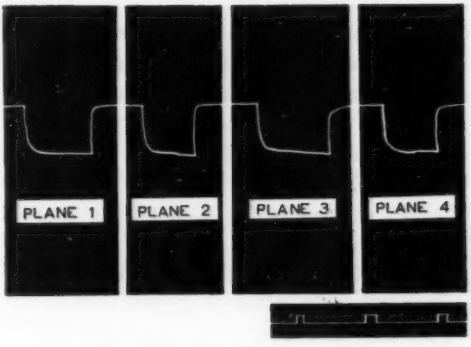

FIG. 4

Female, aged 27. Nitrous oxide/ether. Third apparatus. Showing the deflections produced by supramaximal stimulation of the ulnar nerve in third stage anaesthesia.

DISCUSSION

Gross and Cullen found that the contraction of the gastrocnemius muscle elicited in the dog either by intra-arterially injected acetylcholine, or by electrical stimulation of the nerve, is less in dogs anaesthetized with ether, tribromethanol, or thiopentone, than in dogs anaesthetized with cyclopropane or ethylene. This effect of ether has been attributed to a curare-like action. Paton and Zaimis (1952) stated that a muscle partially paralysed with d-tubocurarine cannot maintain tetanus at its initial strength—that is to say it may begin a powerful contraction but this falls off rapidly. If we attribute a curare-like action to ether we might expect a difficulty in maintaining tetanus in the deeper plane of ether anaesthesia.

Clinically there is no doubt that ether does potentiate the action of d-tubocurarine. The results reported here, however, show that even deep anaesthesia with ether, given for short periods, does not produce a peripheral blocking action in man, presumably because the more susceptible parts of the nervous system are affected first. It is possible that a peripheral blocking action would appear if a deep level of anaesthesia were maintained but it was not considered justifiable to test this hypothesis.

There is no evidence of a peripheral blocking action in relation to thiopentone either. It was not possible to test this other than by alteration in the threshold of response to stimulation, and by showing that the muscles could maintain tetanus during anaesthesia. It seems likely that any peripheral blocking action which may

occur in man will appear only in very high dosage.

SUMMARY

Thirty patients were given pethidine/thiopentone/nitrous oxide. Thirty-four patients were given nitrous oxide/ether. All patients were tested with electrical stimuli applied to the ulnar nerve to determine either; (1) the minimal level of stimulation at which a twitch could be elicited in the supplied muscles; or (2) whether tetanus could be maintained after induction of anaesthesia, and at different levels of anaesthesia, where this was applicable.

It was concluded that there was no satisfactory evidence for any peripheral blocking action during the induction of a deep level of ether anaesthesia or during anaesthesia with pethidine/thiopentone/nitrous oxide.

ACKNOWLEDGMENTS

I wish to acknowledge my indebtedness to Dr. H. H. Pinkerton of the Department of Anaesthetics, Western Infirmary, for his encouragement; to Dr. A. G. Miller, also of the Department, for his ready co-operation; to Dr. R. Taylor for a helpful suggestion incorporated in the apparatus finally evolved. I am indebted to Professor C. F. W. Illingworth for granting me the facilities of the Department of Surgery; to his surgical staff for their forbearance; to his technical staff for their help, including the production of the illustrations. I am indebted to Dr. T. D. M. Roberts of the Institute of Physiology, University of Glasgow, for designing the apparatus shown in figure 1, and for making available the stimulator; to T. E. Hotham and A. Macdonald of the Western Infirmary workshop, for designing the apparatus shown in figure 2, and for making both forms of apparatus.

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Hewer, C. L. (1953). *Recent Advances in Anaesthesia and Analgesia*, 7th ed., p. 13, London: Churchill.

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ADDENDUM

My attention has been drawn to an important paper on this subject by Poulsen and Secher, 1949 (Poulsen, T., and Secher, O., *Acta. Pharmacol.*, **5**, 196). By experimental work on rabbits, Poulsen and Secher show that there is a peripheral curare-like action exerted by ether, that this takes some time to develop, and that the effect is more marked on the response to single shocks than on the response to tetanizing shocks. This might be taken to explain both the clinical impression in some cases of a less vigorous response to single shocks in deep ether anaesthesia, and the absence of a demonstrable effect when tetanizing shocks were applied during the induction of deep anaesthesia.

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TENDON-JERKS AND ANAESTHESIA

BY

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TEXT-BOOKS of anaesthesia seldom mention the effect of general anaesthesia on the tendon-jerks, no doubt because this information has not been relevant in clinical anaesthesia. In the course of other work it became necessary to examine these effects. Tendon-jerks were accordingly tested in seventy-two patients subjected to anaesthesia. The biceps (B), triceps (T), supinator (S), knee (K), and ankle jerk (A) were used. In twenty-eight of these patients only the knee and the ankle jerks were tested. This was because the arms were being used for other purposes. Premedication consisted, in most adults, of papaveratum 1/3 gr (20 mg) and scopolamine 1/150 gr (0.45 mg). In some young or small patients, morphine 1/6–1/8 gr (10.8 mg), and atropine 1/100 gr (0.65 mg) were substituted.

Twenty-eight patients were given nitrous oxide/ether anaesthesia using a semi-closed circuit; 5 litres/minute of nitrous oxide were given with 2 litres/minute of oxygen. Fourteen of these patients were given an intravenous induction with a small dose of sodium thiopentone. The dosage usually varied from 200–300 mg but in two cases doses of 500 mg were used.

Tendon jerks were tested; first in the conscious patients, then in each plane of third stage anaesthesia as it was attained. (The planes were judged clinically. The signs of anaesthesia used were those described by Guedel (1937). It was, of course, appreciated that these signs were described in relation to drop-ether, but they were considered reasonably applicable to the technique in use.) An attempt was made to deepen anaesthesia to a point at which tendon-jerks could not be elicited. In some cases this was not achieved, either because of shortage of time, or because it was not considered justifiable to deepen anaesthesia further.

The other forty-four patients were given pethidine hydrochloride/sodium thiopentone/nitrous oxide; 5 litres/minute of nitrous oxide were given with 1.5 litres/minute of oxygen in semi-closed circuit. Pethidine 50 mg was given intravenously at least five minutes before the induction of anaesthesia. Sodium thiopentone was given intravenously in a dosage of 300–500 mg. Tendon-jerks were tested, first in the conscious patient, and again a few minutes after the beginning of the inhalation of nitrous-oxide/oxygen.

RESULTS

The principal results have been incorporated in tables I and II.

Table I(A) shows the effect of the nitrous oxide/ether technique.

(1) In plane 1, only one patient lost all tendon-jerks. All the patients lost some tendon-jerks.

TABLE I

Observed relation between tendon-jerks and plane of anaesthesia

(A) NITROUS OXIDE/ETHER

No.	Sex	Age	Conscious					Plane 1					Plane 2					Plane 3					Plane 4				
			B	T	S	K	A	B	T	S	K	A	B	T	S	K	A	B	T	S	K	A	B	T	S	K	A
1	F	17	+	+	–	+	+	–	+	–	+	–	–	–	–	–	–										
2	F	17	+	+	–	++	+	+	+	–	+	–	–	–	–	–	–										
3	F	20	+	+	+	+	+	–	–	–	–	–	–	–	–	–	–										
4	F	22	+	+	+	+	+	–	–	–	+	+	–	–	–	+	+	–	–	–	+	+					
5	F	29	+	+	+	+	+	+	–	–	+	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
6	F	30	+	+	+	+	+	–	–	–	+	–	–	–	–	–	–	–	–	–	–	–					
7	F	49	+	+	+	++	+	+	+	–	++	+	+	+	–	++	+	+	–	–	+	+	–	–	–	+	–
8	M	14	+	+	+	+	+	+	+	–	+	+	–	+	–	–	+	–	+	–	–	–	–	–	–	–	–
9	M	20	+	+	+	+	+	–	–	–	+	+	–	–	–	–	–										
10	M	23	+	+	+	+	+	–	+	–	+	+	–	+	–	+	+	–	–	–	–	+					
11	M	31	+	+	+	+	+	+	–	+	+	++	+	–	–	+	++	–	–	–	–	+	–	–	–	–	–
12	M	34	+	+	+	+	+	+	+	–	+	+	+	+	–	+	++	–	+	–	+	++	–	–	–	+	++
13	M	45	+	+	+	+	+	+	+	–	+	–	+	+	–	–	–	–	–	–	–	–	–	–	–	–	–
14	M	46	+	+	–	+	+	+	+	–	+	+	–	–	–	–	–	–	–	–	–	–					

(B) THIOPENTONE/NITROUS OXIDE/ETHER

No.	Sex	Age	Conscious					Plane 1					Plane 2					Plane 3					Plane 4				
			B	T	S	K	A	B	T	S	K	A	B	T	S	K	A	B	T	S	K	A	B	T	S	K	A
15	F	17	+	+	—	+	+	+	+	—	+	+	—	+	—	—	+	—	—	—	—	—					
16	F	17	++	+	—	++	+	+	+	—	—	—	—	—	—	—	—										
17	F	22	+	+	+	+	+	+	—	—	—	+	—	—	—	—	+	—	—	—	—	—					
18	F	27	+	+	+	++	+	—	—	—	—	—											(1)				
19	F	28	+	+	—	+	+	—	+	—	—	+	—	+	—	—	—										
20	F	43	+	+	+	+	+	+	+	—	+	+	—	—	—	+	+	—	—	—	—	+					
21	F	47	+	+	+	+	+	+	—	—	—	+	—	—	—	—	—										
22	M	17	+	+	—	+	+	+	+	—	+	+	+	+	—	+	+	+	—	—	—	—	—	—	—	—	—
23	M	19	+	+	+	++	++	+	+	—	++	++	—	—	—	+	+	—	—	—	—	—					
24	M	21	++	++	++	++	++	+	—	—	++	++	—	—	—	+	+	—	—	—	+	+	(2)				
25	M	32	+	+	+	+	+	—	+	—	+	+	—	—	—	—	—										
26	M	34	+	+	+	+	+	—	+	—	+	+	—	+	—	+	+	—	—	—	+	+	—	—	—	—	—
27	M	43	+	+	+	+	+	+	+	—	+	+	—	+	—	+	+	—	—	—	—	—					
28	M	43	+	+	—	+	+	—	+	—	—	+	—	+	—	—	+	—	—	—	—	—					

B = Biceps jerk
T = Triceps jerk
S = Supinator jerk
K = Knee jerk
A = Ankle jerk

(1) Tendon-jerks present after thiopentone.
(2) Knee and ankle clonus until Plane 2.
+ = Present
++ = Exaggerated
— = Absent

(2) Six of the thirteen patients who still retained tendon-jerks in plane 1 lost them in plane 2.

(3) None of the seven patients who retained tendon-jerks in plane 2 lost them in plane 3.

(4) Two of the six patients who still retained tendon-jerks in plane 3 are known to have lost them in plane 4.

In case No. 12, the ankle jerk became persistently exaggerated in second plane anaesthesia.

In four patients, anaesthesia was not deepened sufficiently to abolish all tendon-jerks. In early fourth plane anaesthesia one patient retained active knee-jerks and another patient retained knee and ankle-jerks.

Table I(B) shows the effect of the thiopentone/nitrous oxide/ether technique.

(1) In plane 1, only one patient lost all tendon-jerks. Eleven additional patients lost some tendon-jerks.

(2) Three of the thirteen patients who retained tendon-jerks in plane 1 lost them in plane 2.

(3) Five of the ten patients who retained tendon-jerks in plane 2 lost them in plane 3.

(4) Two of the four patients known to

TABLE II

Observed relation between tendon-jerks and anaesthesia

			Conscious					Unconscious				
No.	Sex	Age	B	T	S	K	A	B	T	S	K	A
29	F	17	+	+	+	+	+	+	+	+	+	+
30	F	19	+	+	+	+	+	+	+	—	+	+
31	F	23	+	+	+	+	+	+	+	+	++	+
32	F	28	+	+	+	+	+	+	—	+	+	+
33	F	30	+	+	+	+	+	+	+	+	+	+
34	F	39	++	++	++	++	++	++	++	++	++	++
35	F	51	+	+	+	++	+	+	+	+	++	+
36	M	17	+	+	—	+	+	+	+	—	+	+
37	M	18	+	+	+	+	+	+	+	+	+	+
38	M	23	—	—	—	—	+	—	—	—	—	+
39	M	28	+	+	+	+	+	+	+	+	+	+
40	M	32	+	+	—	+	+	+	+	—	+	+
41	M	41	++	+	+	++	++	+	—	—	++	++
42	M	48	+	+	+	++	+	+	+	—	++	+

B = Biceps jerk
T = Triceps jerk
S = Supinator jerk
K = Knee jerk
A = Ankle jerk

\+ = Present
++ = Exaggerated
— = Absent

retain tendon-jerks in plane 3 lost them in plane 4.

In three patients, anaesthesia was not deepened sufficiently to abolish all tendon-jerks.

Table II shows the effect of the pethidine/thiopentone/nitrous oxide technique. Fourteen of the patients given this combination were chosen so as to match in sex and approximate age the individual patients of the ether series.

In ten of the fourteen patients it was possible, when the patient was anaesthetized, to elicit all those tendon-jerks which had been present when the patient was conscious. No patient lost more than two of the five jerks tested. It may also be noted that case No. 32 developed hyperactive knee-jerks after induction of anaesthesia.

In the remaining twenty-eight patients given pethidine / thiopentone / nitrous oxide, only the knee and the ankle-jerks were tested. All the patients who showed both knee jerks in the conscious state retained them both when anaesthetized, except for one patient who lost his knee-jerk after induction of anaesthesia. Other points of note in this supplementary series are:

(1) In one patient, it was not possible to elicit either the knee or the ankle-jerk, before or during anaesthesia.

(2) In another patient, it was possible to elicit the knee-jerk, but not the ankle-jerk, before and during anaesthesia.

(3) Three patients developed ankle clonus after induction of anaesthesia.

The impression derived from the whole series is that the supinator-jerk is more easily abolished than any other, and that the knee and the ankle-jerks are on the whole, retained later than the others.

DISCUSSION

The patients given ether were to some extent selected, in that patients with pre-existing respiratory disease were excluded. The two series of patients given ether are matched in sex and fairly closely in age. The patients in the pethidine series shown in table II were chosen to match the patients in the ether series as closely as possible in age and sex.

We have, therefore, three comparable series of patients subjected to different techniques of anaesthesia. The patients in table I(B) differ from the patients in table I(A) in that they received thiopentone as an addition to the anaesthetic. The patients in table II differ from the patients in table I(B) by the substitution of pethidine for ether. (They also received, on average, slightly more thiopentone and a slightly lower percentage of oxygen.)

Comparing tables I(A) and I(B), the only difference seems to be that the patients given thiopentone tend not to retain their tendon-jerks for as long as the patients not given thiopentone. In such a short series, of course, this slight difference could easily have arisen by chance. Only a very striking difference would be worth considering.

The patients in table II were at a stable level of light anaesthesia giving signs consistent with the first or second plane of third stage anaesthesia. Comparing tables I and II the difference is very marked. With the ether techniques, the tendon-jerks may or may not be affected in light anaesthesia; the effect is clearly variable and unpredictable. With the

pethidine technique, any effect on the tendon-jerks is very rare.

A tendon-jerk is a type of spinal-reflex. The reflex arc consists of a sensory nerve originating in muscle-spindles, and a motor nerve originating in anterior horn cells in the appropriate segment of the spinal cord. These anterior horn cells are subjected to influence by a complicated system of facilitatory and inhibitory descending tracts. These include facilitatory cortico-spinal pyramidal fibres from area 4 of the cortex (Magoun and Rhines, 1946), cortico-reticulospinal inhibitory fibres from the area 4-S of the cortex (Magoun and Rhines, 1946; Niemer and Magoun, 1947), and reticulo-spinal facilitatory fibres (Rhines and Magoun, 1946). The reflex-arc on whose integrity the tendon-jerk depends may, therefore, be interrupted by structural or functional injury to the afferent nerve, the efferent nerve and neuromuscular junction, or the anterior horn cell; or the response may be changed by altering the behaviour of cells in the cerebral cortex, in the reticular formation of the medulla or in other parts of the central nervous system which are responsible for facilitatory and inhibitory influences playing on the anterior horn cells.

At what point, then, does general anaesthesia affect tendon-jerks? It is generally assumed that the depressant effects of general anaesthetics are made manifest on the various parts of the nervous system in the order of their oxygen requirements and therefore in inverse order to the survival-time (found in animals) of nerve-cells of the several parts of the nervous system subjected to anoxia (Hewer, 1953). On this assumption the peripheral nerves would be the last part of the nervous system to be affected by anaesthetics. This is confirmed by animal work carried out by Heinbecker and Bartley (1940). These authors compared the action of ether and pentobarbitone sodium on the various parts of the nervous system of the frog, turtle and cat. They found that the relative susceptibility of the parts of the nervous system varied, and that the speed of anaesthetic action also varied, occurring in the order: cortex; spinal cord; basal ganglia and brain stem; peripheral portions of the sympathetic and somatic nervous systems.

The action of ether on the tendon-jerks seems, therefore, more probably to be due to a central action than to a peripheral action. It is, however, difficult to exclude the possibility of a direct action on the muscle spindles forming the sensory receptors for the reflex arc involved. This theory would attribute a considerable range of variation in the susceptibility to ether of these sensory receptors, so that in different patients they may be affected at very different levels of anaesthesia. Variations in susceptibility may also be found in the type of muscle-spindle involved in the particular reflex tested. All the findings of this study could be explained on this basis.

It was much more easy to test the suggestion that the effect of ether on the tendon-jerks might be due to a peripheral motor block. Brown (1955) has shown that there is no significant peripheral blocking action of the somatic motor system during the induction of a deep level of ether anaesthesia in man. Tendon-jerks are, however, frequently abolished in light ether anaesthesia. It follows that

this abolition is not caused by any peripheral blocking action of ether on the somatic motor system.

If, therefore, the effects of ether on the tendon-jerks are due to an effect on the motor side of the reflex-arc, then these effects must occur at the anterior horn cells of the spinal cord, or in connection with cells in the cerebral cortex giving rise to tracts descending to the anterior horn cells. The occasional appearance in third stage anaesthesia of enhancement of tendon-jerks might be attributed to an imbalance of effect among the various facilitatory and inhibitory systems of nerve-fibres impinging on the anterior horn cells of the spinal cord.

SUMMARY

Tendon-jerks were tested in seventy-two patients subjected to anaesthesia. Three techniques of anaesthesia were used: nitrous oxide/ether; thiopentone/nitrous oxide/ether; and pethidine/thiopentone/nitrous oxide. Patients given ether usually, though not always, lost all tendon-jerks in the deeper planes of anaesthesia. In some patients given ether, the tendon-jerks were lost early in anaesthesia. The result was not easily predictable. Patients given pethidine/thiopentone/nitrous oxide seldom lost tendon-jerks. The site at which ether exerts its effects on the tendon-jerks was discussed.

ACKNOWLEDGMENTS

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THE MECHANISMS CONCERNED IN THE ABNORMAL PROLONGATION OF EFFECT FOLLOWING SUXAMETHONIUM ADMINISTRATION, WITH REFERENCE TO THE USE OF NEOSTIGMINE

BY

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SINCE the introduction of suxamethonium into clinical anaesthesia many cases of unexpected prolongation of paralysis have been reported.

Often, however, much essential information is lacking and the exact mechanism responsible for the abnormal effects remains obscure; in consequence the treatment of these patients has been uncertain, hazardous, and in some cases frankly detrimental.

It is now recognized that animal experiments may have little bearing on the effects of neuromuscular blocking agents in different species. Furthermore with suxamethonium the normal transient effects of the drug, together with the low incidence of abnormal reactions, severely limits the results that may be obtained by experimentation with human volunteers. Therefore only further careful and accurate recording of all abnormal responses that occur in clinical anaesthesia will lead to a fuller understanding of neuromuscular blocking mechanisms in man.

In this paper a case is reported showing some unusual and hitherto unreported features. The mechanisms which may be concerned in the unexpected prolongation of effect following the administration of suxamethonium are reviewed. Some clinical and theoretical aspects and the use of neostigmine in treatment are discussed.

MECHANISMS INVOLVED IN ABNORMAL RESPONSES

There are several theoretical mechanisms by which abnormal prolongation of effect can follow the use of suxamethonium (table I).

Type 1. *Pseudopotentiation.*

Central respiratory depression followed by either hyperventilation and acapnia, or hypoventilation and carbon dioxide retention, can produce apnoea or imperfect return of respiratory movement which may be mistakenly attributed to the relaxant used. Reflex laryngeal apnoea also occurs. There is no absolute evidence that in man suxamethonium has a central depressant action, but such a possibility cannot be excluded in the light of present knowledge; it is suggestive that patients seem to retain no memory of the incidence reported. Electrical stimulation of main nerve trunks may help to differentiate central depression from true myoneural block (Churchill-Davidson, 1955).

Type 2. *Real overdose.*

Owing to rapid hydrolysis of suxamethonium in the presence of normal pseudocholinesterase level, increasing the dose fourfold only doubles the duration of effect (Bourne, 1953). With real overdose, therefore, apnoea is not unduly prolonged and terminates abruptly in a normal characteristic manner. Thus the initial impression was gained that accurate dosage was " neither possible nor necessary ". Both conceptions are incorrect and may lead to difficulties.

Type 3. *Relative overdose.*

Absolute overdose may be of no consequence in the presence of a normal pseudocholinesterase level, but should this level be depressed then even moderate dosage, if uncontrolled, can lead to prolonged paralysis, particularly when repeated doses are given or continuous intravenous infusion is used. Even with gross pseudocholinesterase depression, however, abnormal effects can be avoided, and provided the dose of suxamethonium is small and controlled the paralysis is not necessarily abnormally prolonged. (See case report.)

Type 4. *Paralysis by succinylmonocholine.*

Suxamethonium (succinyldicholine) is hydrolysed by way of succinylmonocholine; this derivative was originally thought to be without action. Should the accumulation of the monocholine breakdown product reach the critical level necessary, however, it produces a depolarizing myoneural blocking action slower in onset, less complete, and longer lasting than the dicholine, with marked accumulative effect (Foldes and Tsuji, 1953) and in some respects more like decamethonium (Ellis, Wunck, Fanelli, and de Beer, 1953). Such paralysis might persist after repeated or continuous administration of suxamethonium.

Type 5. *Abnormal antidepolarizing (" curare-like ") action.*

Work by Paton and Zaimis (1952) led to the classification of the relaxants into two groups—antidepolarizing or competitive inhibitors (" curare-like "), and depolarizing (suxamethonium and decamethonium) according to the conception of the type of neuromuscular block produced. The absolute distinction between the two blocks was difficult (Bovet, Bovet-Nitti, Guarino, Longo and Fusco, 1951), and it was suggested that compounds possessing both actions might exist. Later Foldes (1954) suggested that there were two main classifications, the type of block produced being dependent upon the ability of the drug to change the configuration of the receptor proteins of the endplate in the species under consideration. Hunter (1954) considered that all myoneural blocking agents possessed the same properties in varying degrees but differed mainly in the amount of over-activity they produced. These latter classifications endeavour to explain results which show that in the muscles of several mammalian species decamethonium and suxamethonium are not purely depolarizing (Zaimis, 1953; Hall and Parkes, 1953; Brand, 1952; Philippot and Dellamagne, 1953). Though the initial paralysis is due to depolarization, possibly by " changing their grip " the drugs later act as antidepolarizing agents. With decamethonium such

an atypical action may occur in man due to an altered response of the motor end plate in the clinically weak muscles of myasthenia gravis (Churchill-Davidson and Richardson, 1953). It appears that these atypical actions occur more readily with large doses of the drugs. It is unconfirmed whether suxamethonium (or its derivative succinylmonocholine) has such a "dual" action in myasthenia (or perhaps in some apparently normal individuals), but in the present state of our knowledge such a possibility cannot be excluded.

Type 6. Electrolyte imbalance.

Abnormality of the blood potassium, hypocalcaemia and changes in the ionic composition of the extracellular fluid may cause abnormal responses to relaxants, but these are not regarded as common occurrences in the ordinary course of clinical anaesthesia and will not be discussed further here.

It should be noted, however, that all types of potentiation may be intermingled in one patient and that occurrence of one may "trigger" or aggravate potentiation of another type.

Modifying effect of pseudocholinesterase depression.

Though a low pseudocholinesterase level may be the main factor present in relative (type 3) overdose, such depression may also modify the other responses; the depolarizing monocholine paralysis (type 4) should more readily occur, and would be potentiated. However, with an antidepolarizing block such as occurs in type 5, a low pseudocholinesterase (plasma cholinesterase) level would, *per se*, have

TABLE I

Types, clinical signs, and causes of prolonged neuromuscular blocks which may follow suxamethonium

Types	Clinically	Cause
Frank overdose	Very slightly prolonged apnoea —no residual paralysis	Simple overdose
Relative overdose	Slightly prolonged or prolonged apnoea—perhaps transient residual paralysis	Cholinesterase depression
Monocholine paralysis	Persistent residual paralysis following prolonged administration	Breakdown products reaching critical level
Antidepolarizing paralysis	Persistent paralysis following single, or repeated doses	Altered response of neuromuscular junction

TABLE II

The effect of cholinesterase depression and neostigmine administration on neuromuscular blocks

Type of neuromuscular block	Effect of cholinesterase depression		Effect of neostigmine	
	Pseudo-ChE. (plasma)	True ChE. (muscle)	Anti-ChE.	Direct activity(?)
Depolarizing	Potentiates	Nil	Potentiates	Muscle stimulant
Anti-depolarizing	Nil	Resistance	Reverses	Muscle stimulant

little influence because the true cholinesterase of the myoneural junction is usually more constant than the plasma enzyme (Marchand, 1952); should a coincidental depression of the true cholinesterase occur, a resistance to paralysis would be expected (table II).

THE USE OF NEOSTIGMINE IN PROLONGED PARALYSIS

The administration of neostigmine in clinical anaesthesia has hitherto been restricted to the application of the drug as an antidote to the antidepolarizing relaxants. Neostigmine should potentiate the depolarizing paralysis following suxamethonium; and it does so in the types 2, 3 and 4 described. The apparently paradoxical reversal of "depolarizing" paralysis in cases reported (Grant, 1952; Ruddell, 1952; Hodges, 1953; Cowan, 1954; Argent, Dinnick, and Hobbiger, 1955) suggests that a "dual" paralysis (type 5) is a clinical possibility in man following suxamethonium—perhaps due to subclinical changes at a myoneural junction in certain individuals.

Before making this assumption, however, a further possible mechanism must be considered. The effectiveness of neostigmine in decurarization is allegedly due to an anticholinesterase action. However, more powerful anticholinesterases may be less effective than neostigmine in decurarization and it has been suggested that anticholinesterase activity is not an important factor, the effect of neostigmine being due to a specific muscle stimulant action at the muscle endplate (Riker and Wescoe, 1950; Wescoe and Riker, 1951). Though this action may be of minor importance it does not preclude the possibility that the effect of neostigmine is produced by mechanisms other than anticholinesterase activity (Smith, Cohen, Pelikan and Unna, 1952); These may be of value in both antidepolarizing and depolarizing blocks.

CASE REPORT

(F. H., August 1952)

A healthy man aged 42 had a severe secondary haemorrhage after an appendicectomy, necessitating laparotomy and a transfusion. Post-operative pulmonary and abdominal infection which resisted antibiotic therapy led to an empyema. Rapid deterioration in the patient's condition suggested a *B. Coli* septicaemia with a subphrenic abscess, and exploration was planned.

Anaesthetic management.

After premedication with atropine 1/150 grain a sleep dose of thiopentone was followed by nitrous oxide-oxygen. Suxamethonium (total 100 mg) was given by two or three intermittent injections during the operative procedure which lasted 70 minutes. Post-operatively, only weak diaphragmatic respirations returned with tracheal tug and no further improvement occurred. Three and a half hours from the induction of anaesthesia, the patient was still completely flaccid, but he did not appear to be deeply anaesthetized and feeble finger movements and wrinkling of the face occurred. No response of any kind followed large doses of nikethamide.

A venous blood sample was obtained for pseudocholinesterase estimation. In the light of previous experience (Hodges, 1953) it was felt that though a depolarizing relaxant had been administered neostigmine might reverse the condition; 1.5 mg in 10 ml of water was given slowly. An obvious improvement in respiratory excursions followed, the patient immediately responded vaguely to commands by voluntary limb movements, and in half an hour, four and a half hours from the induction of anaesthesia, he was fully conscious and rational. Surprisingly, he had no recollection of his experience.

A grossly abnormal pseudocholinesterase level was confirmed (10 micromols/ml/hour: normal 130 to 310—method of de la Heurga, Yesinick, and Popper, 1952). After a slow but steady recovery he was fit for discharge, but was readmitted two months later with acute small bowel obstruction. Pre-operative estimation showed his pseudocholinesterase to be still 50 per cent below the lower level of normality. After premedication with pantopon 1/6 grain (10 mg) and scopolamine 1/300 grain (0.22 mg), thiopentone 250 mg nitrous oxide-oxygen (65–35 per cent) and intermittent pethidine were administered. Relaxation was cautiously induced and maintained with a 0.1 per cent suxamethonium drip. When adequate relaxation was obtained the patient was rendered apnoeic by controlled respiration through an absorber, but spontaneous respirations were allowed from time to time; this was considered essential to act as a guide to the rate of infusion. During the three-hour operation the total dose of pethidine was 150 mg and of relaxant

250 mg (90 mg/hr). Full spontaneous respirations returned within two minutes of stopping the infusion and the patient regained consciousness on extubation. His recovery was uneventful and he remains healthy to the time of this report, thirty months later.

DISCUSSION

Several important features are illustrated by the clinical findings.

This case demonstrates that the pseudocholinesterase can become depressed in serious illness in the absence of liver disease, anaemia, and malnutrition, factors which are generally accepted as being associated with such depression. At the time of his prolonged paralysis his pseudocholinesterase level was grossly depressed when, in spite of severe illness, his nutrition and haemoglobin level had been maintained. The serum albumin was the only one of the liver function tests to show depression. Serial estimations over two months showed that the slow recovery of the pseudo-cholinesterase ran closely parallel with the clinical state (fig. 1). This association has been discussed elsewhere (Hodges and Harkness, 1954).

The result of this case seems to justify the opinion that a low pseudocholinesterase level is no contra-indication to the rational and safe use of suxamethonium.

The paralysis in the first administration was abnormally long even allowing for a grossly depressed pseudocholinesterase. The second administration shows that a man with severe cholinesterase depression

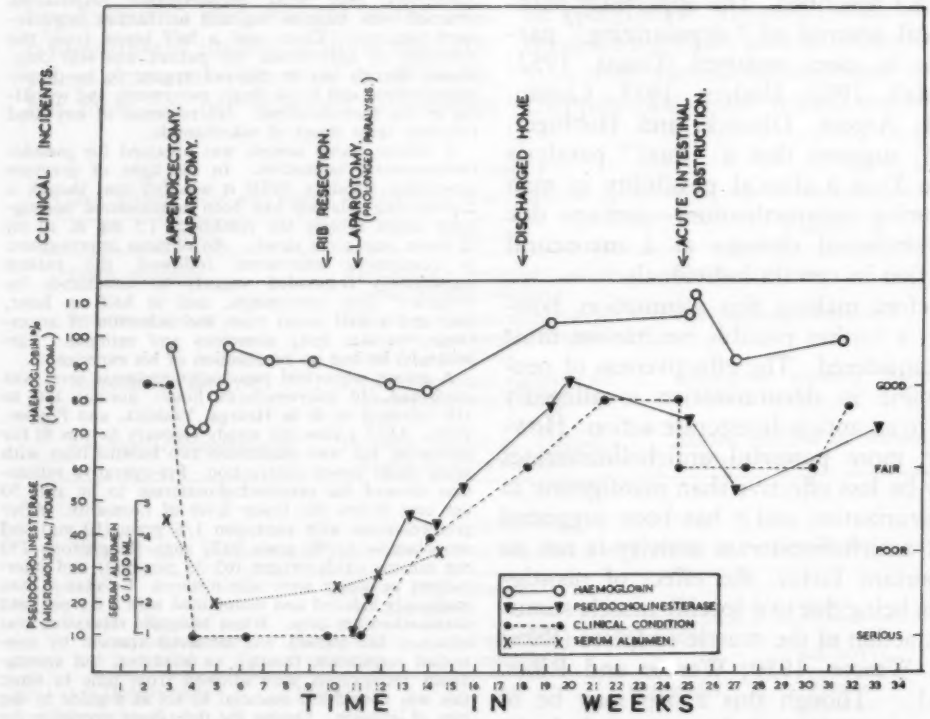

FIG. 1

is quite capable of successfully hydrolysing, redistributing, or eliminating suxamethonium when given slowly. This not only confirms the theoretical assertions that even a 50 per cent reduction in enzyme level need not by itself prolong the effect of suxamethonium unduly (Thesleff, 1952; Argent *et al.*, 1955) but also the opinion that a low level *per se* is no contra-indication to the administration of the drug (Hodges and Harkness, 1954).

The immediate recovery from the effect of suxamethonium administered during the major anaesthetic (when 250 mg produced three hours paralysis) suggests that the effects following the first administration (when 100 mg produced four and a half hours paralysis) was not solely due to simple overdose. Likewise the freedom from untoward effect in the second administration shows that the breakdown products were also eliminated and suggests that the paralysis during the first administration was not due to succinylmonocholine. Furthermore, the reversal by neostigmine, unless it was due to a very marked direct stimulatory action, suggests that a depolarizing paralysis (types 3 and 4) was not present.

Though there is as yet no positive evidence that suxamethonium can have an antidepolarizing action in normal man, I suggest that the evidence strongly supports the possibility that such a block occurred in the present case; the beneficial effects of neostigmine being due to the typical decurarizing anticholinesterase action. It could be that the predisposing factor was the method of administration and not the total dose. It is possible that a sensitive individual may be quite capable of dealing with suxamethonium in a drip infusion—the plasma enzyme being fully employed during the whole administration and redistribution from the neuromuscular junction playing little part

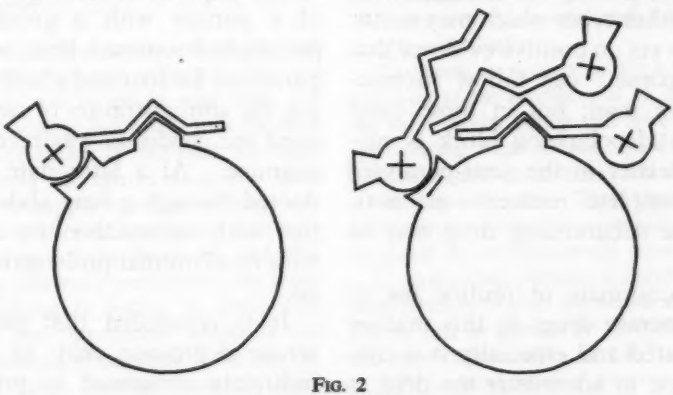

FIG. 2

True Cholinesterase

Both the negatively charged choline-combining grouping and the ester-splitting grouping are combined with one choline ester molecule.

Substrate excess. The choline ester molecules compete for the active groupings of the enzyme molecule. Half combinations result, on which the enzyme is unable to act.

From "Pseudocholinesterase", *St. Bartholomew's Hospital Medical Journal*, February, 1954—with permission of the Editors.

—yet when a similar, or lesser, total dosage is given by intermittent injection the depleted plasma level may not prevent a relative flooding of the neuromuscular junction within the first seconds after each injection. Thereafter only very rapid redistribution can allow the plasma to play any further part in the elimination. This flooding of the strategic site might not only completely inhibit the true cholinesterase responsible for the local hydrolysis (Lehmann, 1954—see figure 2) but might be the factor predisposing to the "dual" response which has been shown in susceptible species and individuals to take place more readily with heavy dosage.

CONCLUSIONS

Pseudocholinesterase depression is not the only factor concerned in prolonging paralysis due to suxamethonium, but such depression may predispose to or aggravate paralysis of other types which may occur.

There is as yet no positive evidence that a "dual response" can follow suxamethonium in man; but in those cases where an antidepolarizing block is suspected—especially in the semi-paralysed state of incomplete recovery—an anticholinesterase decurarizing drug may be of value.

The indiscriminate or routine use of anti-cholinesterase drugs in this manner is not advocated and especially it is considered unwise to administer the drug to the completely apnoeic patient. Though neostigmine has been used successfully, Tensilon, because of its more transient action, is probably a wiser choice.

With suxamethonium elementary precautions against total overdose and sudden flooding should be taken—best achieved by drip infusion. In long cases respirations should be allowed to return, from time to time as fully as surgical conditions allow and the drip regulated to compensate for any tendency to sluggish recovery.

In the present state of our knowledge the exact mechanisms at work in any individual case may be obscure, making reversal difficult, maybe hazardous, and perhaps impossible; nevertheless the rational use of dilute solutions allows a high degree of control and safety not only in the normal, but also in sensitive individuals.

SUMMARY

Several possible types of paralysis following suxamethonium are formulated and discussed, together with the mode of action of neostigmine and its possible beneficial effects in these cases.

The paper is illustrated with the report of a patient with a grossly depressed pseudocholinesterase level who remained paralysed for four and a half hours following the administration of suxamethonium until the condition was reversed by neostigmine. At a later date he was conducted through a long abdominal operation with suxamethonium as a relaxant with no abnormal prolongation of paralysis.

It is concluded that pseudocholinesterase depression may be directly and indirectly concerned in prolongation of paralysis but there is evidence that other mechanisms may be involved, including atypical antidepolarizing blocks. For this reason anticholinesterases may be of value in specific instances.

The method of administration and not

the total dose of suxamethonium administered may be the causative factor in some cases, and certain precautions are essential. Abnormal effects are least likely to occur with dilute controlled dosage.

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BOOK REVIEW

Chirurgie Réparatrice (2e Série). By R. Merle d'Aubigné. Published by Expansion Scientifique Française.

One of the great difficulties about writing a book is to decide whom you are writing it for, who will read it, and how to ensure that what you write is reasonably up to date by the time the book is published. The author of this book has made his decisions firmly and carried them into effect exactly. He is writing for experienced surgeons: he will be read by all who have to do with conditions mainly of an orthopaedic nature, and what he has written is extremely pertinent to the problems and procedures of today. It deals with arthritis of the hip; ligamentous lesions of the knee and consolidation of fractures of the leg; the neuropathies of tabes; brachial paralysis; non-union of the carpal scaphoid; Dupuytrens contracture; mal-united Colles fractures; surgery of the intervertebral disc, as well as some problems of anaesthesia for patients with rheumatoid arthritis, and other short related contributions.

The list of contributors is impressive, but the work of Professor Merle d'Aubigné of course predominates. Consequently the style is clear, the illustrations are excellent, the approach is objective and the careful and critical analysis of results is invaluable.

The book is written in French, but its construction of the sentences is so simple that even the most insular Briton will have no difficulty in appreciating what is said. Just in case this is optimistic, the author has attached to each section a summary in English. These summaries are clear and concise and the few examples of quaint word or phrase do not obscure the sense.

It seems that perhaps a decision might have been taken as to what the author should be called. On the title he appears as " P Merle d'Aubigné ", and later more usually as " R " and occasionally as " Pr ". And since a reviewer must criticize, " Rheumatoid " (arthritis) is spelt with an " e " before the " u ". But it can be taken as an indication of the pleasure in reading and the value of possessing this book, that such trivialities should have to be picked out for mention.

Bryan McFarland

Brit. J. Anaesth. (1955), 27, 492

UNUSUAL STENOSIS OF TRACHEA IN AN ADULT

BY

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HAVING chosen after due consideration a cuffed endotracheal tube, and having achieved the accepted conditions of anaesthesia and relaxation for its introduction, the anaesthetist normally and rightly expects to achieve his objective without much further trouble. After passing the bevelled end through the glottis, the further progress of the tube is usually easy unless an error has been made in the size of the tube chosen or unless the attachment of the cuff should prove faulty. Eventual failure to introduce a plain, oral tube, two sizes smaller than the original choice, farther than a few centimetres beyond the cords confirms a growing suspicion of possible abnormalities and calls for investigation. We encountered such circumstances in the following case:

The patient was a female, aged 62, of slight build and in only a fair state of health. She had for many years suffered from dysuria following a plastic repair for prolapse of the uterus. The difficulty had been treated with some success by repeated dilatations of the urethra, but eventually the stricture became intractable. Intravenous pyelography showed considerable dilatation of the renal system and it was decided to perform a bilateral transplantation of the ureters. Considerable oedema of the tissues with dilatation of the ureters was found at the operation, which was performed without surgical incident. from this operation the patient made an immediate satisfactory recovery, but an ascending infection resulted in a fatal pyelonephritis on the tenth postoperative day. This unhappy outcome permitted us to complete an account of the anaesthesia with a detail which would not otherwise have been possible. The autopsy afforded an opportunity to examine more fully this patient's trachea and so explain the early difficulty which was encountered.

Anaesthesia. After premedication with morphine 1/6 grain (10 mg) and atropine 1/100 gr. (0.6 mg), and following the intravenous administration over five minutes of pethidine hydrochloride 60 mg, anaesthesia was induced with thiopentone 0.4 g. The injection of d-tubocurarine, 18 mg, associated with the inhalation of nitrous oxide and oxygen, provided suitable conditions for intubation under direct vision. A No. 10 Magill cuffed tube appeared at once to be too large and intubation was now attempted with a No. 9 cuffed tube. This tube entered the glottis but failed to continue its progress for more than 2–3 cm. A second attempt produced the same result and, blaming the attachment of the cuff for the difficulty, a further change was made to a No. 8 plain tube. This, of course, passed the glottis easily, only to be held up at the same distance within the trachea. Accepting the probability now of some tracheal obstruction, the intubation was abandoned lest worse befall. A Guedel airway was inserted and the anaesthesia was maintained with nitrous oxide and oxygen, further small doses of pethidine and d-tubocurarine being exhibited as required *via* an intravenous drip.

At the close of the operation, sufficient anaesthesia and relaxation were maintained to allow of bronchoscopy in an attempt to discover the cause of the failure to intubate. The 11-mm blade of the bronchoscope passed the glottis easily, thus confirming that the choice of a No. 9 Magill tube for the second attempt was not unreasonable. The external diameter of a No. 9 tube is stated by Gillespie (1948) to be 11¼ mm and for a No. 8 10⅓ mm. About 2–3 cm beyond the glottis the trachea began to narrow, the bronchoscope was held up and further progress was not attempted. No evidence of tumour or bulging inwards of any portion of the upper trachea could be made out nor was any trace of bleeding visible. A general stenosis of the trachea appeared to be present and to be the explanation of the obstruction. No respiratory complication developed in the early postoperative days and our resolve to call upon radiologist and laryngologist at a later date became unnecessary in the eventual outcome of the case.

Autopsy (with relation to examination of the trachea). "The trachea contains a small quantity of viscid, purulent material. Immediately below the vocal cords, at the beginning of the trachea, the circumference measures 3.5 cm; 4 cm below this the circumference measures 2.4 cm and this narrowed segment persists for a length of 2.5 cm. Immediately above the carina the circumference measures 3.4 cm."

Thus the tracheal diameters at the points mentioned are 11.1 mm, 7.6 mm,

and again 10.8 mm. Had the trachea maintained its maximum diameter throughout, the No. 8 tube would certainly have passed easily, and indeed allowing for the normal effect of the absence of the cartilages in the posterior tracheal wall, the 11-mm bronchoscope could have been introduced to the carina. According to the scale of measurements tabulated by Gillespie (1948), this trachea could only have accommodated a No. 3 tube, or at most a No. 4 Magill tube. The circumstances suggested that this patient had a congenital stenosis of the trachea and that this was an uncommon condition. Further investigation was to confirm that such was the case.

DISCUSSION

It was natural that the first question should be put to the pathologists—in their view such a stenosis was very unusual. A visit to the anatomists revealed that normally and usually the trachea was of uniform bore, certainly in the adult. Reference to a standard text-book of anatomy (Gray, 1950) indicates a uniform tracheal diameter. Further reference to abnormalities of the trachea as detailed in Schwalbe (1912) suggests that a gradual stenosis such as was encountered in this case is most uncommon. Indeed such narrowing as is described in this article was invariably associated with deformities of the tracheal or laryngeal cartilages or with abnormal origins of the bronchi. The cases described include, with one exception, only infants, none of whom survived. The exceptional case was a man of 70 years who had a combined stenosis of larynx and trachea. Study of an article by Evans (1949) on congenital obstruction of the respiratory tract indicates that stenosis is associated with various malformations of the tracheal cartilages. He states further that the normal infantile trachea is usually of uniform calibre but in some instances local narrowing may apparently be due to cartilaginous softening, with the exception of the reference already mentioned (Schwalbe), attempts to find a description of a similar instance in an adult have been unsuccessful. Holinger *et al.* (1950) describes various types of congenital stenosis but these again were in infants and produced early symptoms of their presence. Similarly, reference to Carruthers (1951) and Martin and Albright (1947) leads to a description of laryngeal and tracheal stenosis quite dissimilar to that presently encountered. Thomson and Negus (1948) in their classical treatise give a full account of the aetiology of stenosis of the trachea, as do Jackson and Jackson (1934) in their manual on bronchoscopy. This case could only be reasonably included under congenital abnormalities according to their classifications. No case similar to this presentation is described unless clearly due to compression from some demonstrable extrinsic cause. No malformation or abnormal origin of bronchi was present in our case, and we appear to be dealing with a patient whose trachea suffered from a lack of uniform development. The deficiency over the middle third was insufficient to cause any reported inconvenience in its use as an air-passage, and the gradual narrowing and re-expansion of the lumen presented no opportunity for the lodging of secretions which might have given rise to some infective process.

Anaesthetists commonly foresee that

deviations from normal in the trachea are to be expected and that they may present some problem in intubation. Here we had an unexpected difficulty due to a condition of the trachea acknowledged by anatomists and pathologists to be extremely unusual. Our problem might have been much more acute had we felt that a cuffed endotracheal tube was a necessary part of the anaesthetic technique. The unusual explanation for the failure to complete an apparently routine intubation is an excuse for this short publication. The photograph (fig. 1) shows the extent of the stenosed segment of the trachea.

FIG. 1

The extent of the stenosed segment is indicated by the approximation of the cut edges of the tracheal wall.

SUMMARY

Failure to introduce an endotracheal tube of suitable size more than a short distance beyond the cords aroused much curiosity. The explanation proved interesting in that the trachea showed no evidence of disease (intrinsic or extrinsic) but revealed an unusual narrowing in its middle third which was presumably due to some incomplete early development.

ACKNOWLEDGMENTS

We wish to express our thanks to Mr. W. S. Mack (Urologist to the Western Infirmary) for permission to describe this case, and to Dr. J. H. Stephens (Pathology Department, Western Infirmary) for his help and interest. We are indebted to Mr. Kerr for the photograph.

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Brit. J. Anaesth. (1955), **27**, 495

SOME OBSERVATIONS ON THE USE OF THE HEWER MATTRESS

BY

E. HACKER

St. Andrew's Hospital, London

IN 1953 C. Langton Hewer devised a new mattress to be used on operating tables. With this mattress it is possible to place a patient safely and securely in Trendelenberg position without shoulder and other supports, and also without the necessity of breaking the table at the foot end to transfer some of the weight from the shoulders (or pelvis) to the lower legs.

In his paper, Hewer describes the disadvantages and dangers inherent in all previous methods of securing a patient in this position.

As these difficulties were well known, to surgeons and anaesthetists alike, the new mattress found widespread acceptance and appears in increasing numbers of operating theatres. According to Hewer, a patient in steep head-down tilt will rest securely on an operating table if his skin is in direct contact with a mattress the upper surface of which is corrugated, as the skin, if dry, will adhere to the rubber with sufficient force to prevent any material sliding movement.

In other words, the patient has to be moved and placed without bed sheet and gown on the theatre table.

The difficulty of lifting a heavy and adipose patient who is naked is obvious to anybody who has at any time witnessed or given a helping hand to the performance.

Hewer also designed three small rubber cushions with corrugated surfaces which for various reasons enhance the merit of the mattress. One of these cushions is placed under the nape of the neck, one under the lumbar spine, and one under the Achilles tendons of the patient. When they are used—as they should be—it is noted (*ibid.*) that the slight sliding movement of the patient as described by Hewer does not occur.

If we accept this as a fact, and in addition presume some adherence of the skin to the Hewer mattress (even though covered with a hospital bed sheet) we find that a patient placed on the table with this sheet between him and the corrugated mattress, will lie as securely on it as he would with his skin in direct contact with the rubber, and no sliding movement takes place even in steep Trendelenberg tilt. Therefore there is no need for the often difficult manoeuvre of moving a naked patient from the trolley to the table and *vice versa*. The patient can be left in his usual theatre gown, lifted quite easily by his bed sheet, placed on the table covered with the Hewer mattress and its three rubber cushions, and can be tilted safely to the required degree of head-down position.

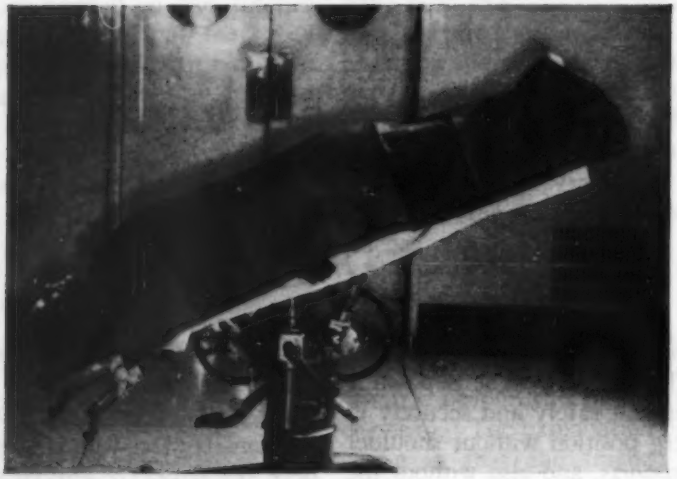


FIG. 1

The accompanying photograph (fig. 1) demonstrates the method here described.

The theatre staff are pleased with this new manoeuvre, they find it easy, simple and expeditious. I am grateful to Mr. J. East, F.R.C.S., for the illustration.

REFERENCE

Hewer, C. L. (1953), *Lancet*, **1**, 522.

SOCIETY NEWS

THE SHEFFIELD AND EAST MIDLANDS SOCIETY OF ANAESTHETISTS

THE eighth Annual General Meeting of the Sheffield and East Midlands Society of Anaesthetists was held at the County Hospital, Lincoln, on Saturday, May 23, 1955. The following office bearers were elected:

President: Dr. H. E. Pooler (Chesterfield).
Vice-President: Dr. J. R. Spark (Nottingham).
Treasurer: Dr. J. P. Hope (Sheffield).
Secretary: Dr. C. A. S. Hamilton (Sheffield).
Members of Council: Dr. W. M. Dinwoodie (Derby)
Dr. J. Johnston (Sheffield)
Dr. F. Logan Turner (Leicester)

Dr. W. J. Patterson, retiring President, delivered his address, "As Others See Us", which was greatly appreciated by the large gathering present.

The meeting was concluded with very hospitable entertainment by the Lincoln Anaesthetists followed by an excellent meal served in the Staff dining-room by the courtesy of Matron.

PROGRAMME

1955

November 5: Annual Dinner, to be held at George Hotel, Nottingham.
Speaker: Professor W. W. Mushin.

December 3: Dr. P. D. Bedford. "Effects of Anaesthesia on the Aged" (at Sheffield).

1956

March 23: Registrar's Paper, to be read at Derby.

May 12: Annual General Meeting, to be held at Leicester.
Speaker: Mr. L. B. Wevill, F.R.C.S.

Brit. J. Anaesth. (1955), **27**, 497

A THERMOCOUPLE FOR THE BRAIN

BY

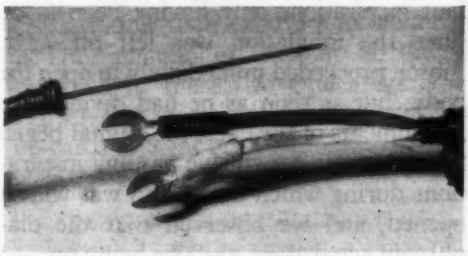
J. D. WHITBY

Newcastle General Hospital

IT seems ludicrous that the reading of a rectal thermometer should be taken as a guide for deciding how long a major cerebral artery may be safely clamped by a neurosurgeon. Although the temperature of the brain must approximate that of the blood passing through it, temperature gradients are present throughout the body during hypothermia and the reading on the thermometer will vary with the distance that it is inserted.

There are no thermometers on the market which are suitable for recording brain temperatures, and it seemed that the simplest way of making a thermocouple for this purpose would be by adapting a brain cannula. Mr. W. E. Robinson of the Cambridge Instrument Company has very kindly done this for me.

The lower $1\frac{1}{2}$ cm of the cannula were sacrificed. A copper-constantan thermocouple was threaded in and the tip welded in and rounded off, leaving the sensitive point flush with the surface. The rubber covered lead is 15 feet long and the instrument is used with a standard Cambridge skin temperature outfit. Sterilization is by autoclaving.



Parkinson, Jensen and Vars (1954) found that in dogs the brain would tolerate much lower temperatures than the heart, and it should be possible to reduce the temperature of the brain below that of the heart by local cooling, although some general cooling will probably always be necessary.

The arterial method that they used is hardly applicable to man, and although the internal carotid arteries pass close to the naso-pharynx it would be difficult to apply effective cooling to that confined space. The ventricular cavities do not offer much hope either, but there does not seem to be any reason why the surface of the brain should not be irrigated with cold saline.

REFERENCE

Parkinson, W. M., Jensen, J. M., and Vars, H. M. (1954). *Ann. Surg.*, **140**, 284.

ON CHLOROFORM AND OTHER ANÆSTHETICS

THEIR ACTION AND ADMINISTRATION

BY

JOHN SNOW, M.D.

Licentiate of the Royal College of Physicians

(*Continued from page* 467)

In the Experiments 24 and 25, related above, the animals were first made insensible by vapour of moderate strength, in order to keep them quiet for the application of the stethoscope whilst the stronger vapour was inhaled, but when animals are made to breathe air containing ten per cent. or so of vapour of chloroform from the first, death takes place in the same manner, *i.e.*, by the direct action of the chloroform on the heart. Under these circumstances the creatures die suddenly, after a brief interval of agitation and excitement, without previous insensibility. This is what has happened in some of the accidents from chloroform to be related further on; whilst in many cases death has happened after a longer or shorter period of insensibility.

In many of the fatal cases of inhalation of chloroform the alarming symptoms commenced whilst the patient was still inhaling, but in a considerable number there was no sign of danger until after the inhalation had been left off; and this is a result which may be imitated on animals. The two following experiments, which were made at Dr. Sibson's on December 27th, 1849, illustrate this point.

Experiment 29. The subject of this experiment was a young guineapig, about half grown. Ten or twelve drops of chloroform were put into a short bent metal tube, lined with bibulous paper, which formed part of an inhaler, and one end of the tube was applied to the animal's nose, which closed it. The guineapig consequently breathed backwards and forwards through the tube, which was, moreover, warm from being held in the hand. It struggled at first, endeavouring to get away from Dr. Sibson, who held it; but in twenty or thirty seconds it became quiet; and observing that the conjunctiva was insensible, I withdrew the chloroform, and the guineapig was laid on the table. We remarked that the breathing was very slow; and scarcely had this remark been made, when it ceased altogether, three or four inspirations only having been made since the inhalation was left off. Dr. Sibson proceeded immediately to open the body; but as soon as he had divided the integuments of the chest the animal began to make some convulsive gasping inspirations during which the mouth was widely opened, and we observed that the diaphragm acted alone, the ribs being pressed inwards by the atmosphere. The heart was not contracting at all when the pericardium was first opened; but in a few moments the auricles began to contract

rhythmically. The lungs were of a light red colour.

Experiment 30. Another guineapig of the same age was treated in the same manner, except that I removed the tube from the nose for a short time between every two or three inspirations, in order to see if I could not make it insensible in this way without a fatal result. In half a minute or so the animal no longer required to be held, but lay on the table without making any resistance, the conjunctiva being still sensible, and some motion of the limbs, apparently voluntary, remaining. It only inhaled once after Dr. Sibson ceased to hold it, and whilst in the state just described, and then only for two or three inspirations. After this it appeared quite insensible, and no more chloroform was given. It breathed at first naturally, except more slowly and gently than before the inhalation; but the breathing became slower, and ceased altogether about a quarter of a minute after the inhalation had been left off. The body was opened immediately. When the pericardium was opened, the auricles were observed to contract slightly, and the contractions increased after their exposure to the air.

The amount of vapour in the air breathed by these two guineapigs is not known, but there is no doubt that it was upwards of eight per cent.

FATAL CASES OF INHALATION OF CHLOROFORM

If it were possible for a medical man to mistake or disregard the symptoms of approaching danger, and to go on exhibiting vapour of chloroform, diluted to a proper strength, till the death of the patient, this event would take place slowly and gradually, as in Experiment 23, related above, and every other experiment in which the air did not contain more than five per cent. of vapour. The action of the heart would survive the respiration; there would be a great tendency to spontaneous recovery, and the patient would be easily restored by artificial respiration, if it were performed whilst the heart was still acting; as I have always found it to be successful in animals under these circumstances.

In examining the recorded cases of fatal inhalation of chloroform, we shall find, however, that they have none of them taken place in this gradual manner; but that in all cases the fatal symptoms, if not the actual death, have come on very suddenly. Dr. Sibson was, I believe, the first to point out that, in the fatal cases of inhalation of chloroform, death was caused by its paralysing the heart; but he was not at that time aware of the physical conditions under which this agent may act directly on the heart. In commenting on the first four deaths from chloroform, he says: * " In all the four cases it is manifest that the immediate cause of the instantaneous death lay in the heart. The heart, influenced by the poison, ceased to contract, not from the cessation of respiration, for the heart in asphyxia will beat from one to three minutes after respiration has ceased, but from immediate death of the heart. There is no doubt a combination of causes operating to destroy the heart's contractile power: the mental influence the congestion in the systemic, and that in

* London Medical Gazette, vol. xlii, 1848, p. 109.

the pulmonary capillaries, will all have a material influence." And further on he says: "We are obliged, then, from the experience of these cases, to conclude, that in man the death is usually instantaneous, and due, as every instantaneous death is, to paralysis of the heart. In animals the death is usually due to paralysis of the muscles of respiration. It is chiefly owing to the superior control of the mind over the body in man, that in him the poison acts on the heart more than in dogs."

I had been long aware that chloroform was capable of paralysing the heart in animals; and, indeed, that sulphuric ether would do so when it could reach the heart in sufficient quantity;* and soon after Dr. Sibson's remarks I was able to ascertain and point out the strength of the vapour which will produce this effect, and how one may avoid the risk of it, by having the vapour sufficiently diluted.† The greater number of experimenters who have killed animals with chloroform have found that the action of the heart continued after the breathing ceased; but they did not either control or ascertain the proportion which the vapours of chloroform bore to the inspired air. In Mr. Thomas Wakley's experiments the action of the heart continued after the breathing had ceased; and this was the case in a great number of experiments performed by a Commission which reported to the Society of Emulation of Paris, in 1855.‡

This Commission came to the conclusion that, in all instances in which animals are killed by chloroform, the action of the heart survives the respiration; but they might have administered chloroform to an equal number of human patients without any one of them being cut off by sudden paralysis of the heart. If animals were usually to die suddenly of paralysis of the heart, when the chloroform is given in a manner similar to what may be called its ordinary administration to patients, we should be at a loss to know how this agent could be used at all. It is only reasonable to suppose that, in experiments so conducted, that mode of death should usually be met with which would occur to the human subject, if the chloroform were continued, in the disregard of dangerous symptoms, till death should ensue. I have, indeed, been informed of several instances in which animals died in a sudden, and what was thought an unaccountable manner, whilst chloroform was given to prevent the pain and struggles which would be occasioned by physiological experiments. In these cases there is no doubt the heart was paralysed; but the experimenters were often too intent on other matters to observe the circumstance. By a proper arrangement of circumstances, however, one may produce at will the event in animals which occurs, in fact, so very rarely to a patient, although it may at any time happen if great caution, guided also by right principle, is not used.

Air, when saturated only at 60° Fah., contains 12 per cent of vapour of chloroform, and at 70° 19 per cent; and 8 or 10 per cent. in the inspired air is capable, as we have seen, of causing sudden death by paralysing the heart; but in practice the air is usually far from being saturated, in passing over a handkerchief or similar material, even at the temperature at which it is inhaled; and this is

* On the Inhalation of the Vapour of Ether, p. 82.
† Medical Gazette, vol. xlii, p. 415.
‡ See l'Union Médicale, 1855, No. 13.

generally lowered considerably by the absorption of caloric by the chloroform, as it changes its condition from a liquid to a vapour. Moreover, air strongly charged with vapour of chloroform is not easy to breathe, owing to its pungency; and the physiological knowledge and close attention of every medical man who administers this medicine causes him to withdraw it immediately on the least appearance of danger.

Case 1. The first death from chloroform was that of Hannah Greener, which occurred at Winlaton, near Newcastle, on the 28th of January, 1848. The patient was a girl of 15, who required to have the nail of the great toe removed. A similar operation had been performed on the other foot, in the previous November in the Newcastle Infirmary, when ether was administered with a satisfactory result. The following is the account of the accident by Dr. Meggison, who administered the chloroform: "She appeared to dread the operation, and fretted a good deal: in fact, she commenced sobbing on our entering the house, and continued so until seated in the operating chair, and commencing the inhalation, which was done from a handkerchief on which a teaspoonful of chloroform had been poured. After drawing her breath twice, she pulled my hand from her mouth. I told her to put her hands on her knees, and breathe quietly, which she did. In about half a minute, seeing no change in breathing, or alteration of pulse, I lifted her arm, which I found rigid. I looked at the pupil and pinched her cheek and, finding her insensible, requested Mr. Lloyd to begin the operation. At the termination of the semilunar incision she gave a kick or twitch, which caused me to think the chloroform had not sufficient effect. I was proceeding to apply more to the handkerchief, when her lips, which had been previously of a good colour, became suddenly blanched, and she spluttered at the mouth, as if in epilepsy. I threw down the handkerchief, dashed cold water in her face, and gave her some internally, followed by brandy, without, however, the least effect, not the slightest attempt at a rally being made. We laid her on the floor, opened a vein in her arm, and the jugular vein, but no blood flowed. The whole process of inhalation, operation, venesection, and death, could not, I should say, have occupied more than two minutes."*

An examination of the body was made the day after death by Sir John Fife and Dr. Glover, who reported as follows:— "The body was that of a well-grown female of about fifteen years of age. . . . On opening the chest, the lungs were not collapsed. One or two very slight adhesions were encountered on separating them from the walls of the chest. The external appearance of both lungs, over the whole surface, but especially in the inferior portions, was that of organs in a very high state of congestion. They were mottled with patches of a deep purple, blueish, or scarlet hue. They were everywhere crepitant. Along the outer and interior border of both lungs, particularly of the upper lobe of the left lung, were several emphysematous bubbles of small size. . . . The pulmonary tissue was filled with bloody froth, which was also found

* London Medical Gazette, vol. xli. p. 255.

in the interior of the bronchi, mixed with mucus. There was no appearance of hepatization. On examining the larynx and trachea, the epiglottis was found reddened at the summit, of a vermilion hue. The mucous membrane of the larynx was redder than natural—mottled with vascular patches. The sinuses of the larynx contained a good deal of dark mucus. The œsophagus was healthy. The stomach was distended with food. Some of the veins were more distinct than usual. The heart contained dark fluid blood in both its cavities: very little in the left. Its structure, and that of the great vessels near it, quite healthy. The brain, externally and internally, was more congested than usual; and the ventricles contained rather more than the usual quantity of serum."*

Sir John Fife and Dr. Glover expressed the opinion that chloroform caused death by producing congestion of the lungs. After the further experience which has been gained respecting chloroform, this opinion of the mode in which it caused death, in the above case, would probably not now be offered. Indeed, in similar cases which have since occurred, the same opinion has not been advanced and as the lungs were crepitant throughout, in the above case, it is probable that the congestion was not greater than is commonly met with in cases of sudden death without haemorrhage. Dr. Simpson published his opinion, immediately after this case occurred, that it was not caused by the chloroform, but by the brandy which was given when the alarming symptoms came on; but Dr. Meggison replied that only a teaspoon of brandy was given; that it was swallowed, though with difficulty; that there were no symptoms of choking from it; and that the girl was without pulse when it was given.†

From the lips becoming suddenly blanched in the above case, there is every reason to conclude that the heart was suddenly paralysed. The patient breathed for a little time after this, and was able to swallow, though with difficulty. The process of inhalation occupied only a little more than half a minute in Dr. Meggison's estimation; yet he appeared to think that the fluid drachm of chloroform which he had put on the handkerchief had evaporated; for he was about to apply more when the alarming symptoms appeared. But supposing only one-third of the drachm was actually inhaled into the lungs in the half minute, the vapour would necessarily be in a highly concentrated state—probably twenty cubic inches of vapour in 200 cubic inches of air, which would be adequate to occasion the result which happened. In the short space of about half a minute, the blood could not be uniformly impregnated with the vapour; only a portion of it could pass through the lungs in the time; yet, as the patient was rendered insensible, this portion must necessarily have been highly charged with vapour.

* Opus cit., p. 253.

† Opus cit., p. 341.

(*To be continued*)

WORLD CONGRESS OF ANAESTHESIOLOGISTS, 1955

THERE will be unanimous agreement among all, whatever their nationality or the relative profundity of their interest in anaesthesia, that this first World Congress of Anaesthesiologists held at Scheveningen was an enormous success. From the stately opening in the Hall of Knights (Ridderzaal) at the Hague to those final impressive moments in the Central Sessions Hall of the Kurhaus when the World Federation of Societies of Anaesthesiologists was founded, those attending were kept busy, interested and entertained. The Great Hall of the Ridderzaal, an ancient 13th-century castle, where the Queen of the Netherlands annually carries out the state opening of her Parliament, with its vast beamed roof and candelabra, reminded many of the great hall at Lincoln's Inn where the Association of Anaesthetists of Great Britain and Ireland held its Dinner in 1946 to celebrate the centenary of the discovery of Ether Anaesthesia. Here the Congress was formerly declared open by Mr. J. G. Suurhoff, Her Majesty's Minister of Social Affairs and Public Health. In his opening remarks Mr. Suurhoff drew attention to the wide scope of this specialty. Advances in anaesthesia had not only led to tremendous advances in surgery, particularly of the heart and lungs, but the skill and knowledge of the practitioners in this specialty had been applied to useful purpose in the treatment of poliomyelitis and to assist in the modern treatment of the pyschiatric patient.

Mr. Suurhoff was followed by Mr. Zwyndrecht an Alderman of the Hague who welcomed the delegates on behalf of the Burgomaster and Corporation. Prof. Dr. E. Grzegorzewski of the World Health Organization, which had taken the greatest interest in the planning for the World Congress and World Federation, pointed out that anaesthesia had been and still was a major public problem in many parts of the world. It could be regarded as "the preventive discipline to all branches of medicine where surgical intervention is required". In splendid, truly statesmanlike speeches delivered in four languages Dr. G. R. Ritsema van Eck, President of the Organizing Committee emphasized that all the efforts of delegates and congressioners during the week must be directed to the one end—to serve mankind in the relief of suffering. He particularly welcomed the scientists and surgeons who might be associated with anaesthetists in the various national societies to whom "we owe so much and whom we regard as our greatest friends".

The academic sessions of the Congress reached a very high standard in several instances. They were held in the afternoons in the large Central Hall in the Kurhaus at Scheveningen and in the mornings the delegates divided into two groups to attend papers and discussions in the two smaller North and South Session Halls. A special delight at these sessions were the beautiful floral decorations of

the stage and rostra, and the excellent arrangement that had been made for translation.

On each chair were headphones through which, by the simple adjustment of a switch the words of the speaker could be heard in English, French and German. Although (not surprisingly) the translators had a little difficulty with some of the more technical papers, the arrangements worked on the whole splendidly and added very greatly to the success of the Congress.

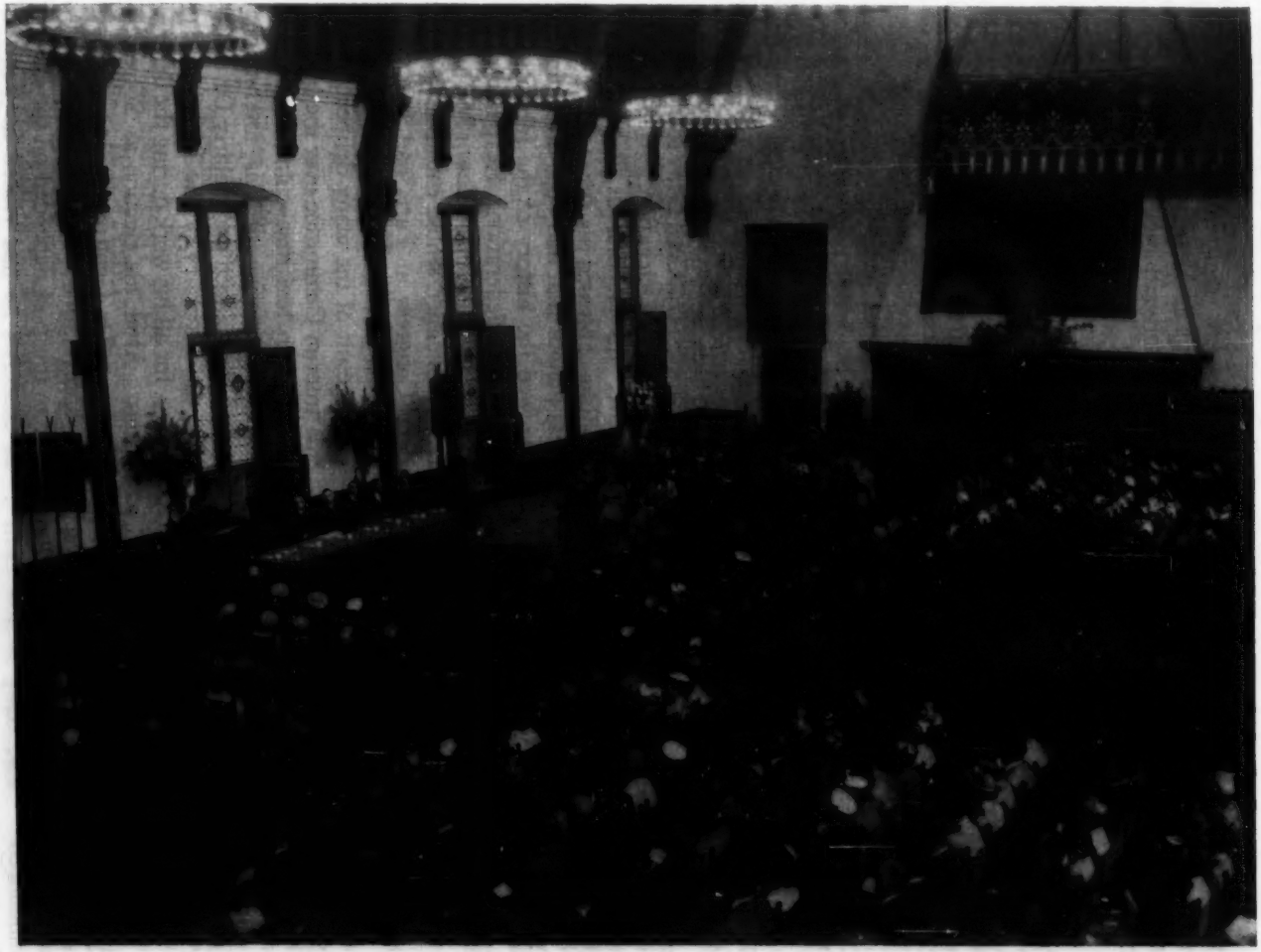
The speakers had been allotted various times for their papers which were strictly enforced. This occasionally led to some embarrassment and, perhaps more unfortunately, to readers hurrying unduly through communications. In one case an interesting paper was "cut off", after about one-third of its course through a misunderstanding. These incidents, however, were the price, and probably a very small one, which had to be paid for the necessary limitation of the more voluble speakers.

It was to be expected that the main themes of this gathering would be Hypothermia, Hypotension, and the Relaxant Drugs. It would be quite invidious to draw attention to any few of the many communications given, but the programme of papers listed below shows the wide variety of the fare offered. Certainly they demonstrated the contrasting approaches made to the problems of anaesthesia.

Not the least interesting features were the three "Panel-Discussions". At these, questions previously written in were answered by a panel of four experts on the subject under consideration, presided over by a moderator. One recalls with pleasure Professor Pask's lucid answers and deft dealing with an obscure question on carbon dioxide and also the eloquence of Professor Beecher. It was clear that all present did not agree with the latter's pronouncement that the only two advances in anaesthesia in the last two decades which had permitted the surgeon to do what had hitherto been impossible were Hypotension and Hypothermia.

The social side of the Congress was well up to the standard of its more serious aspects. The weather on the whole was excellent except for one morning and afternoon of rain, and the admirable bathing facilities were greatly appreciated. Each afternoon there was an excursion for Associate Members to the various attractive and historically interesting features in the neighbourhood. In addition there was a dance and variety show in the Hotel Witte Burg in the Hague, one feature of which was attractive folk dancing in national costume. This was an evening of complete relaxation and conviviality which will long be remembered by those present. The closing Congress Dinner was held at the Palace Hotel, Scheveningen.

The climax of this outstanding Congress was the General Assembly of the various national delegates and of the Congressioners in the Central Hall of the Kurhaus at which was formed the World Federation of National Societies of Anaesthesiologists. On the stage delegates from the majority of countries in the world sat on either side of Dr. Harold Griffiths, who had been the chairman of the Interim Committee responsible for the planning of the World Federation. Dr. Goldblat (Belgium), Secretary of the Interim Com-



The Opening of the International Congress, 1955, in the "Ridderzaal" at the Hague.

mittee, then gave the report of the Committee on Credentials to the Assembly, which had approved of the credentials of the following delegates:

Delegates

Argentine: Dr. M. Alarcon, Dr. G. Varela
Australia: Dr. N. James
Austria: Dr. O. Mayrhofer
Belgium: Dr. A. Goldblat
Brazil: Dr. O. Bastos
Canada: Dr. H. R. Griffiths, Dr. R. A. Gordon
Chile: Dr. L. C. Guarderas
Columbia: Dr. G. Delgado
Cuba: Dr. Corbello
Denmark: Dr. O. Secher
Finland: Dr. E. O. Turpeinen
France: Dr. J. Boureau, Dr. P. Jaquenard
Germany: Dr. R. Frey
Great Britain: Dr. W. Low, Dr. G. Organe, Dr. J. Gillies, Dr. R. Shackleton
India: Prof. S. Talwalkar
Israel: Dr. Foldes
Italy: Prof. E. Ciocatto
Netherlands: Dr. C. Ritsema van Eck
Norway: Dr. I. Lund
Portugal: Dr. E. Lopes-Soares
South Africa: Dr. Roberts
Sweden: Dr. T. Gordh
Switzerland: Dr. K. Zimmermann
Spain: Dr. D. Monton
Uruguay: Dr. A. Canellas
Venezuela: Dr. C. Hogi

Observers

Czechoslovakia: Dr. H. Kadlic
Greece: Dr. S. Couremenos
New Zealand: Dr. A. Slater
U.S.A.: Dr. L. Wright
U.S.S.R.: Dr. Zeitzen
Yugoslavia: Dr. A. Longhino

The Constitution and Bye-laws as drawn up and approved by the Interim Committee were then unanimously adopted, after which Dr. H. Griffiths announced: "The baby is now born". For legal reasons this Constitution and Bye-laws are registered in the Dutch language. The Nominations Committee then put forward their nominations for the World Federation which were as follows:

President

Professor H. R. GRIFFITHS (Canada)

Vice-Presidents

Dr. C. RITSEMA VAN ECK (Holland)
Dr. A. GOLDBLAT (Belgium)
Dr. R. FREY (Germany)
Dr. CURBELLO (Cuba)

Secretary-Treasurer

Dr. GEOFFREY ORGANE (Great Britain)

Owing to the fact that the Secretary-Treasurer could no longer serve as a National Delegate, being supranational in his duties, Dr. Cecil Gray took his place in the British Delegation.

After his election as President, Dr. Griffiths expressed his appreciation of the great honour that had been done him and reviewed the history of the formation of this World Federation. He paid tribute to the inspiration of Professor Monot and Dr. Thalheimer, whose enthusiasm at the 1951 Congress in Paris had led to the formation of the Interim Committee. It was a result of the many deliberations of this Interim Committee over the last four years which had now culminated in the present formation of this international body. He thanked particularly Dr. Goldblat, the Secretary of the Interim Committee, for all his work during the deliberations. After his election as Secretary-Treasurer, Dr. Geoffrey Organe expressed his appreciation of the honour. If his title to this post depended on the number of countries he had visited he was well qualified, as since the war he had travelled

to fourteen countries in four continents. He said he regarded war as "the greatest futility", and considered it was always the result of misunderstanding. The best way to avoid misunderstanding was to get to know each other better and he was sure that the World Federation had an important part to play in this respect. It was not the purpose of the new Federation to interfere in the affairs of any country or of any National Society of Anaesthesiologists. It would be its duty to give help when help was required and in this he would serve to the best of his ability. He intended as soon as it was possible to visit the member countries to get to know them better. He paid tribute to Dr. Goldblat who had been assiduous in his capacity as Secretary of the Interim Committee.

The election of the Executive Committee followed and the following delegates were elected:

Dr. J. Boureau
Dr. E. Ciocatto
Dr. J. Gillies
Dr. A. Goldblat
Dr. A. Gonzales Varela
Dr. T. Gordh
Dr. R. A. Gordon
Dr. H. R. Griffith
Dr. N. R. James
Dr. O. Mayrhofer
Dr. R. P. W. Shackleton
Dr. Zairo Vieira
Dr. S. G. Talwalkar (co-opted)
Dr. Ritsema van Eck (co-opted)

It was unanimously agreed to send a telegram to Her Majesty the Queen of the Netherlands, expressing the gratitude and loyalty of those present for having so generously given her patronage to the Congress.

The time and place of the next International Congress were left for an early decision by the Executive Committee.

LIST OF LECTURES

Physiology

Prof. Dr. T. Sjøstrand, Stockholm: "Blood Volume and Regulation of Circulation."

Prof. E. A. Pask, Newcastle upon Tyne: "Technique for Assessing the Central Respiratory Depression in the Presence of Muscle Relaxants."

Dr. G. Rodewald, Hamburg, Dr. R. Beer, Marburg: "Haemoxytensiometry."

Dr. R. F. Woolmer, Bristol: "The Role of the Anaesthetist in the Treatment of Bulbar Poliomyelitis and Tetanus."

Dr. H. C. Churchill-Davidson, London: "Acute Massive Pulmonary Collapse."

Dr. P. R. Bromage, Chichester, Portsmouth: "Blood pressure and Vital Capacity."

Dr. R. P. Harbord, Leeds: "Pulmonary Ventilation during Anaesthesia."

Dr. B. Sommer Pedersen, Hillerød: "Anaesthesia and Stress."

Dr. J. D. Robertson, Edinburgh: "Some Effects of Anaesthetics on the Reflex Regulation of the Circulation."

Prof. Dr. P. Knoefel, Louisville: "The Renal Circulation in Anaesthesia."

Dr. J. L. McClung, U.S.N.R., Dr. G. C. Sutton, U.S.N.R., Dr. D. M. Little, U.S.N.R.: "Measurements of the Mechanical and Electrical Events of the Cardiac Cycle; (1) Ether Anaesthesia."

Dr. G. Moricca, Dr. L. Manni, Rome: "Electro- and Balistocardiographic Studies on Patients submitted to Various Types of Anaesthetic Induction."

Dr. Sadi Sun, Aksaray, Istanbul: "Anaesthesia in Intra-thoracic Surgery without CO_2 Absorption (High Flow Technique)."

Dr. H. Göpfert, Heidelberg: "Observations on the Physiology of Respiration in Experiments with 'rapid-action' CO_2 Analysers."

Dr. H. Hörnicke, Hannover, Dr. J. Stoffregen, Heidelberg: "Comparison of Pressure-breathing and Positive-Negative Breathing in Experiments with Animals."

Dr. F. R. Gusterson, Worthing: "Tonsillectomy in Children" (with film).

Dr. G. Arezio, Pavia: "Influence of Glucose-1-phosphate on the modifications of the Electrocardiogram in Anaesthesia."

Dr. D. Carbonera, Dr. V. Freyrie, Pavia: "Respiratory Insufficiencies in Anaesthesia."

Dr. L. Barth, Berlin-Buch: "Left-right Differences during Pulmonary Ventilation in Intermitted Pressure-breathing in the open Thorax."

Dr. H. Oehmig, Dr. J. Stoffregen, Heidelberg: "On a New Apparatus to Stimulate Expectorations."

Dr. D. Mendelsohn, Philadelphia: "Electrocardiographic and Blood Pressure Changes during and following Biliary Tract Surgery."

Dr. S. A. Klein, The Hague: "Some Physiological Considerations on the Administration of Oxygen during General Anaesthesia, especially in the Elderly Patient."

Dr. R. E. Mansfield, London: "The Control of Secretions in Thoracic Surgery."

Dr. B. A. Sellick, London: "The Control of Cardiac Rhythm during Heart-Surgery."

Prof. Dr. A. Quevauviller, Dr. O. Blanpin, Paris: "The Influence of Fluids on Local Anaesthesia."

Artificial Hibernation and Hypothermia.

Dr. P. Huguenard, Paris: "The Protection against Stress by Means of Hypometabolism. Physiology and Methods."

Prof. Dr. E. Ciocatto, Turin: "Physiopathological Studies during Hypothermia in Man."

Prof. R. W. Virtue, Denver: "The Clinical Aspects and the Technique of Hypothermia."

Dr. T. C. Gray, Liverpool: "Metabolic Responses to Trauma Inflicted during Hypothermia."

Dr. B. G. B. Lucas, London: "Hypothermia."

Dr. H. M. Laborit, Paris: "The Notion of a Functional Thermic Optimum in Artificial Hibernation."

Dr. G. Soares Bairao, Dr. A. P. de Almeida, Dr. A. V. Lorenzo, Dr. C. Sawaya, Dr. R. Tenuto, Sao Paulo: "A few Data about our Experience in the Hospital das Clinicas of Sao Paulo with Artificial Hibernation and Neuroplegy."

Prof. H. M. Ausherman, Dr. W. K. Nowill, Dr. S. Boyarsky, Dr. H. E. Hall, Dr. M. Bourgeois-Gavardin, Durham, N. Car.: "Physiologic Variations during Induced Hypothermia in the Dog."

Dr. M. Battezzati, Dr. C. Taddei, Dr. P. Mosetti, Dr. G. B. Gemma, Geneva: "Hypothermia and Extracorporeal Circulation with Exclusion of Heart and Lungs."

Dr. L. H. Wright, New York: "Hypothermia for Cardiac Surgery."

Dr. S. M. H. Holmdahl, Uppsala: "The Tolerance of the Dog under Hypothermia to Respiratory Acidosis."

Dr. L. E. Gelin, Dr. B. Löfström, Lund: "On Peripheral Circulation in deep Hypothermia."

Dr. H. C. Slocum, Dr. C. W. Hughes, Dr. P. Schafer, Dr. T. G. Barilla, Washington, D.C.: "The Influence of Pentobarbital and Thiopental on the Tolerance of the Hypothermic Dog-heart to Ventricular Fibrillation."

Dr. E. Gräfin von Lüttichau, Mannheim: "Induced Hypothermia (Laborit) as an aid in the Treatment of Severest Shock."

Dr. O. H. Just, Berlin: "Clinical Experiences in Extreme Induced Hypothermia."

Dr. J. Cahn, Paris: "Physiology of Artificial Hibernation."

Dr. J. du Cailar, Montpellier: "Artificial Hibernation by means of the Methoniums."

Dr. V. G. Longo, Rome: "Effects of Chlorpromazine on Electroencephalographic and behavioural reactions due to Hypothalmus Stimulations in the Rabbit" (with film).

Dr. J. Thuillier, Paris: "Evaluation of the Analgetics in the Animal by means of Tensiometry" (with film).

Dr. Nicola Cocchia, Dr. Renato Cuoculo, Naples: "Hibernation therapy in Surgery for aortic Transplants."

Dr. J. Bimar, Marseille: "The Combination of Hydergin-Diparcol in Hibernation and potentiated Anaesthesia."

Dr. L. Campan, Toulouse: "Hypersensibility to Hibernotherapy."

Dr. P. Jaquenoud, Dr. J. Mercier, Dr. L. Hartung, Dr. M. Gavan, Marseilles: "Oxygen Consumption and Neuroplegic Drugs."

Dr. C. Horatz, Hamburg: "Hypothermia and Artificial Hibernation."

Dr. G. Hossli, Zurich: "Artificial Respiration (continuous) and Hypothermia in severe Wound Tetanus."

Dr. S. A. Mason, Sanderstead, Surrey: "The Hypotensive Technique and Artificial Hibernation for the Fenestration-operation."

Hypotension

Dr. M. Bozza, Milan: "Resperine as an Aid in controlled Arterial and Cerebral Hypotension in Neurosurgery."

Dr. J. P. Payne, London: "Histamine Release during Controlled Hypotension with Arfonad."

Dr. W. N. Rollason, Hull: "The Electrocardiogram in Hypotensive Anaesthesia."

Dr. G. Carstensen: Würzburg: "Damage to the Kidneys and Anaesthesia."

Dr. B. Haid, Innbruck: "Our experiences with Induced Hypotension during Five Years."

Dr. F. Becker, Würzburg: "Induced Hypotension with Arfonad and Kidney-function."

Dr. P. G. Sironi, Milan: "Observations on hypotensive Activity of Resperine in Anaesthesia for the Micro-surgery of the Ear."

Dr. L. Marogna, Sassari: "Fatal Haemorrhage after potentiated Anaesthesia and Induced Hypotension."

Dr. B. Paradis, Quebec: "Physiological and Clinical Considerations on Induced Hypotension in Anaesthesia."

Dr. M. B. S. Silva Araújo, Oporto: "Ganglioplegics."

Dr. F. M. Meisner-Bülow, Stuttgart: "Induced Hypotension in Haemorrhage" (with film).

Dr. H. C. Voorhoeve, Rijswijk, The Hague: "Controlled Hypotension in Gynaecological Operations" (with film).

Dr. J. Gillies, Edinburgh: "Spinal Block and Hypotension."

Dr. D. E. Hale, Cleveland: "The History and Rational of Induced Hypotension."

Dr. E. Kern, Paris: "Analysis of 600 Observations on Induced Hypotension."

Dr. G. E. H. Enderby, London: "Safety in hypotensive Anaesthesia."

Dr. St. Kubicki, Berlin: "Electro-corticographic Derivations in Hypotension."

Dr. K. Wiemers, Dr. E. Kaniak, Freiburg: "The Influence of Phenothiazines on the Tolerance to Ischaemia of the Rabbit-heart."

Dr. E. Gadermann, Hamburg: "Induced Hypotension in Heart-surgery."

Dr. P. Pellmont, Basle: "The Pharmacology of the Ganglionplegics in Connection with Hypotension."

Free Subjects

Dr. M. D. Leigh, Los Angeles: "Paediatric Anaesthesia."

Prof. Dr. R. A. Hingson, Cleveland: "The Reserve Midget Portable Anaesthesia Machine, Resuscitator and Oxygen Inhalator as the Vehicle for Non-explosive cyclopropane/helium/oxygen Anaesthesia."

Dr. W. Block: Hamburg: "On the Relation of Thiobarbiturates to the Fat Metabolism."

Dr. L. Havers, London: "Controlled Hypotension by Extradural Spinal Block Under Chlorpromazine Cover."

Dr. W. Bakhoum, Dr. S. F. Mankarious, Cairo: "Desoxycortisone in the Treatment of Post-spinal Anaesthesia Headache."

Dr. R. L. Rodriguez-Cantú, Mexico: "Fine Needle Technique in Spinal Anaesthesia."

Dr. H. Ruppert, Berlin: "The Changes in the Motility of the Uterus in Extradural Spinal Anaesthesia."

Dr. G. Hoflehner, Steyr: "Cerebral Damage Caused by Anaesthesia and the Consequences Thereof."

Dr. J. T. Hayward-Butt, Durban, S.A.: "Kemithal Sodium. Review of 50,000 Administrations."

Dr. J. Bark, Tübingen: "Bronchoscopy and Bronchography under Anaesthesia."

Dr. K. L. Schober, Halle: "Experimental Elucidation of the Mode of Action of Peridural Anaesthesia."

Dr. H. Wendl, Hamburg: "Anaesthesia for Caesarean section."

Dr. C. Matteucci, Sassari: "1,000 Epidural Blocks."

Dr. V. Keating, Jamaica: "Anaesthetic Problems in a Tropical Country."

Dr. B. R. Margolius, New York: "The phan tartrate on Respiration during Nitrous-oxide-oxygen Anaesthesia."

Dr. M. Swerdlow, Manchester; Dr. F. F. Foldes, Pittsburg; Dr. L. A. Pirk, New Jersey: "The Use of Nisentil and Levallorphan to Supplement Nitrous-oxide-oxygen Anaesthesia."

Dr. P. Drury Byrne, Dublin: "Modified Electroconvulsive Therapy."

Dr. R. D. Levis, London: "The transference of thiopentone and some Relaxants across the Placenta."

Dr. R. Bryce-Smith, Oxford: "Bronchoscopy for the Anaesthetist."

Dr. N. Guardagni, Los Angeles: "Steroid Anaesthesia in Surgery."

Dr. P. Mundeleer, Brussels: "Anaesthesia in Thoracic Surgery."

Muscle Relaxants

Dr. R. J. Hodges, Portsmouth: "Suxamethonium Tolerance and Pseudocholinesterase Levels in Children."

Dr. J. Bullough, Chislehurst: "Five Years continuous use of Dimethyl-tubocurarine in 5,000 Cases."

Dr. J. K. Harper, London: "The Relaxants in Eye Surgery with special reference to Intra-ocular Manipulations."

Prof. W. W. Mushin, Cardiff: "Further Experimental Studies of Gallamine-triethiodide in Man."

Dr. S. Thesleff, Lund: "The Mode of Neuromuscular-block caused by Acetylcholine, Decamethonium and Succinylcholine."

Dr. A. Vitale, Naples: "The Effect of Muscle relaxation on potassium-metabolism."

Dr. S. Johanson, Dr. W. Hougs, Hellerup: "The Interaction of Benzoquinonium with other Curarizing Substances."

Dr. R. Frey, Heidelberg: "A Short-acting muscle relaxant belonging to the Curare Group. Prestonal."

Dr. H. Brücke, Mürzzuschlag: "A very Active Muscle Relaxant. Imbretil."

Dr. R. Rizzi, Venice: "Muscle Relaxation and Mortality in Surgery."

Dr. T. Gordh, Dr. O. v Dardel, Stockholm: "The Results in a 2-year Period using Succinylcholine Iodide in Anaesthesia."

Dr. S. Schotz, Philadelphia: "Technique of extremely light Analgesia Plus Muscle Relaxants in Major Cardiac Surgery" (with film).

Dr. G. G. Nahas, Paris: "Experimental Defibrilation" (with film).

Dr. R. Brönnimann, Dr. H. Stirnemann, Bern: "Experimental Tetanus by Tetanus-toxin and the Curares."

Dr. N. Cocchio, Dr. Fr. Portolano, Naples: "Tardily absorbable d-Tubocurarine in Anaesthesia."

Dr. R. Brodowsky, Paris; Dr. T. Lada, Lille: "Mediatonal (anaethocurarium) Muscle Relaxant and Potentiating other Curares. Clinical use, Evaluation."

Prof. Dr. M. J. Dallemagne, Liège: "Modes of action of Carnitine and its derivatives on the motor-end-plates and Ganglionic Synapsis."

Dr. M. Hanquet, Liège: "Reciprocal Antagonistic action in Man of the Curaremimetics and Acetylcholinomimetics."

Dr. H. L'Allemand, Giessen: "Haemodynamics of the lung Circulation in diffusion Respiration under Oxygen and under gradual Hypoxia."

Dr. H. Gross, Dr. E. Kepes, Dr. D. Young, Dr. D. Enselberg, New York: "Electrocardiographic changes during anaesthesia for Mitral-commissurotomy."

Dr. E. Lopes-Soares, Dr. C. M. Medres, Dr. R. LaFeria, Lisbon: "Results of 1,500 general anaesthetics with pentothal-tubarine-procaine."

Dr. H. Keszler, Prague: "Pathogenisis and Treatment of Laryngeal Spasm."

Dr. A. G. ElGohary, Cairo: "The Status of Anaesthesia in the Middle East."

Dr. J. Pastorová, Prague: "The Present state of Czechoslovac Anaesthesia."

Dr. M. B. de Oleaga Alarcón, Buenos Aires: "Potentiated Anaesthesia in Endothoracic Surgery."

Prof. D. Bovet, Rome: "Chemical structure and Pharmacodynamic Activities of the Natural and Synthetic Curares."

Prof. W. D. Paton, London: "The Mode of Action of neuro-muscular blocking agents."

Dr. F. F. Foldes, Pittsburgh: "The fate of Muscle Relaxants."

Teaching

Dr. H. Reinhold, Dr. J. v. d. Walle, Brussels: "The Teaching of Anaesthesiology."

Prof. Dr. S. M. Campbell, Toronto: "The Teaching of Anaesthesia in a Canadian University."

Dr. N. R. James, Melbourne: "Basic Principles in the Teaching of Anaesthesia."

Dr. J. G. Fairer, London: "The Use of Visual aids in Teaching the Principles of Anaesthesia."

Prof. H. Griffith, Montreal: "The Teaching of Anaesthesiology."

Dr. E. Wainø Andersen, Dr. W. Dam, Dr. E. Husfeldt, Dr. B. Ibsen, Dr. J. Kirchnoff, Dr. H. Ruben, Dr. O. Secher, Copenhagen: "Teaching and Preliminary Results."

BOOK REVIEW

Erlebtes aus der Thoraxchirurgie (1st edition). By Professor Dr. Rudolph Nissen, Director of the University Surgical Clinic, Basle. Published by Georg Thieme Verlag, Stuttgart. Pp. 65. Price D.M.14.40.

This delightful booklet is the substance of the Mitchell Banks Memorial Lecture on "The Romance of Thoracic Surgery" delivered by the author at the University of Liverpool two years ago. In the space of some 12 pages, Dr. Nissen rapidly flits through the temples of Thoracic Surgery, lighting a small candle at each of the numerous shrines *en route*. The story is a fascinating one, and confirms once again how recent are so many aspects of thoracic surgery, which we take for granted and which have become routine today. He pays but modest tribute to the contributions of modern anaesthesia to this branch of surgery. Mention might well have been made of some of the pioneers of Thoracic Anaesthesia within our own memory and time, and without whom the giants of surgery he describes might well have remained pigmies. Nissen was a former pupil of Sauerbruch and it says much in his objectivity when he speaks of his former chief as follows: "Sauerbruch rejected on principle the idea of setting up anaesthesia as a branch in its own right. . . . His voice was sufficiently influential in the Continent to hold up a development there from which in other countries, thoracic surgery was to benefit more than any other."

The second half of the booklet consists of excellently reproduced portraits, and of now historical and classical articles covering the development of thoracic surgery. British surgeons are well to the fore and portraits and reproductions of papers are shown of Morriston Davies, Henry Souttar and Russell Brock. For those whose German is imperfect a nearly complete translation of the lecture was published in the *American Journal of Surgery*, 1955, **89**, 9.

W. W. Mushin

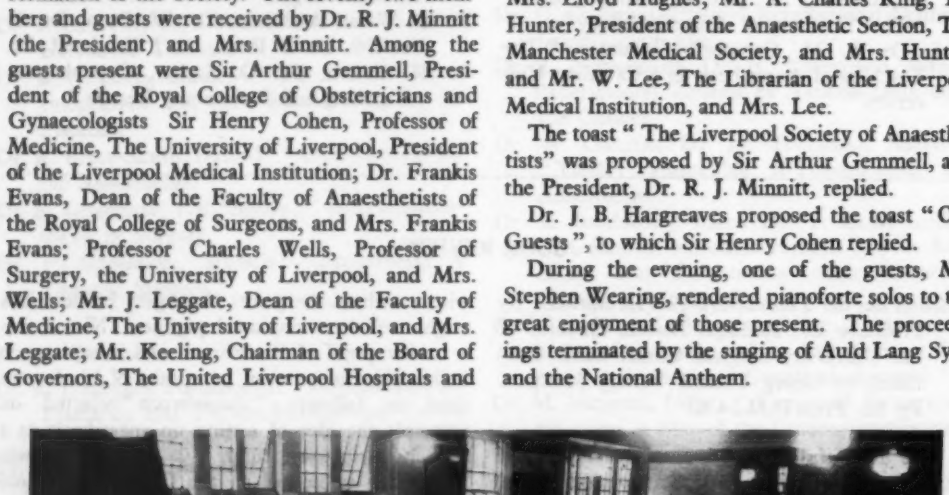
LIVERPOOL SOCIETY OF ANAESTHETISTS

A DINNER was held at the University Club, Mount Pleasant, Liverpool, on Saturday July 16, 1955, to celebrate the twenty-fifth anniversary of the formation of the Society. The seventy-two members and guests were received by Dr. R. J. Minnitt (the President) and Mrs. Minnitt. Among the guests present were Sir Arthur Gemmell, President of the Royal College of Obstetricians and Gynaecologists Sir Henry Cohen, Professor of Medicine, The University of Liverpool, President of the Liverpool Medical Institution; Dr. Frankis Evans, Dean of the Faculty of Anaesthetists of the Royal College of Surgeons, and Mrs. Frankis Evans; Professor Charles Wells, Professor of Surgery, the University of Liverpool, and Mrs. Wells; Mr. J. Leggate, Dean of the Faculty of Medicine, The University of Liverpool, and Mrs. Leggate; Mr. Keeling, Chairman of the Board of Governors, The United Liverpool Hospitals and of the Liverpool Regional Hospital Board, and Mrs. Keeling; Dr. Lloyd Hughes, Chief Medical Officer, Liverpool Regional Hospital Board, and Mrs. Lloyd Hughes; Mr. A. Charles King; Dr. Hunter, President of the Anaesthetic Section, The Manchester Medical Society, and Mrs. Hunter; and Mr. W. Lee, The Librarian of the Liverpool Medical Institution, and Mrs. Lee.

The toast " The Liverpool Society of Anaesthetists" was proposed by Sir Arthur Gemmell, and the President, Dr. R. J. Minnitt, replied.

Dr. J. B. Hargreaves proposed the toast " Our Guests ", to which Sir Henry Cohen replied.

During the evening, one of the guests, Mr. Stephen Wearing, rendered pianoforte solos to the great enjoyment of those present. The proceedings terminated by the singing of Auld Lang Syne and the National Anthem.



A CASE FOR ENDOBRONCHIAL ANAESTHESIA

Sir,—To those of us who are rarely confronted with the problem of anaesthesia for lung operations where the patient is exhibiting large amounts of secretions from the diseased lung, the purchase of something like a Carlens tube seems to be a needless extravagance. In this country such a tube costs around 45 dollars, and such an item might very well lie around in a drawer and rot before we might get to use it, to say nothing of the problem of using it properly when the occasion would demand it.

We recently were confronted with the problem of a patient who had enormous amounts of secretions in his respiratory tract, and who required an emergency operation for which general anaesthesia was deemed necessary. We got by so well that we thought your readers might be interested in how this was handled.

The patient was a 45-year-old white male who for several months had been a diagnostic enigma to both the medical and surgical departments until one day he solved the riddle by proceeding to cough up huge quantities of bile-stained material. It took very little imagination or doing thereupon to prove that what he had started with was an abscess in the liver and that this had succeeded in rupturing through the diaphragm into the chest to find an outlet *via* the right lower lobe bronchus.

By the time he was presented to us this patient had been coughing up in excess of 20 ounces of bile-stained material daily. This would come up in amounts up to an ounce at a time. He had been running a septic fever in spite of all manner of antibiotics, his weight had shrunk to a mere 85 pounds, and his pulse rate hovered around 140 per minute. The proposed operation was an exploration of the liver for an abscess, the surgical approach being through the bed of the 12th rib with the patient in the lateral position right side up. It was felt that a general anaesthesia would be needed for an adequate exploration. An endobronchial technique seemed the only answer to general anaesthesia *sine* inundation.

Having no special endobronchial tubes or blockers, we fashioned an endobronchial technique out of a regular 5/40 Jackson bronchoscope and a No. 8 portex Magill tube. It was found that if one lubricated the outside of the bronchoscope with some petroleum, it would fit the inside of the No. 8 portex tube snugly enough so that they would not move one upon the other during bronchoscope manipulation and yet at the same time it was easy to slide the bronchoscope out once the tube was properly positioned. Thus using a cuffed portex tube and the Jacksonscope we were able to place the tube well into the main stem bronchus on the left under topical anaesthesia and after inflating the cuff we had a perfect fit. For the first time in months the patient breathed freely for about an hour and a half while the surgeons opened a huge abscess in the liver which communicated with a similar one in the right lower lung through a large opening in the diaphragm. At the end the tube was slowly withdrawn as constant suction was being applied with a suction catheter.

It need hardly be pointed out that we were unaccountably lucky to have the left lung available to us. We could not have done the same thing on the right without occluding the right upper lobe bronchus. For those anaesthetists, however, who are fortunate enough to have patients who are considerate enough to allow them to use the left lung for anaesthesia we can recommend this technique very highly.

SEYMOUR SCHOTZ,
Philadelphia, U.S.A.

APNOEA ON CESSATION OF CONTROLLED RESPIRATION

Sir,—In the June issue of the Journal, Dr. Wylie discussed the possible causes of persistent apnoea after prolonged controlled respiration.

I found Dr. Wylie's explanations most interesting, but it appeared to me that for the most part they dealt with persistent apnoea following an overdose, absolute or relative, of anaesthetic agents or muscle relaxants. Manual ventilation for apnoea due to these causes, to my mind, should be referred to as "artificial" rather than controlled respiration. I believe that the term "controlled respiration" should be reserved for that type of respiration in which voluntary respiratory movement has been abolished but where spon-

taneous respiration will resume within a very short period after the anaesthetist ceases to ventilate the patient. The "taking-over" process may be markedly facilitated by deepening anaesthesia and/or by muscle relaxants, but can then easily be maintained for long periods of time with nothing other than nitrous oxide so that it is difficult to see how the initial agents can still be implicated in the final lack of resumption of spontaneous respiration. Yet, this is a condition not infrequently encountered.

I believe that when this delay in resumption of spontaneous respiration following, what I call, controlled respiration occurs it is due to a number of factors, the most important ones being: (*a*) exhaustion of the Hering-Breuer reflex due to persistent over-distension at the neuro-receptors and (*b*) due to the washing-out of carbon dioxide; probably the high oxygen content of the inspired atmosphere plays an associated role here. Such delay of spontaneous respiration is also often seen after use of a mechanical respirator, probably because all three factors are present to an exaggerated degree. It can be avoided by replacing mechanical by manual control toward the end of anaesthesia and by then gradually decreasing its depth until spontaneous respiratory effort resumes.

When respiration has been controlled, as defined above, delayed spontaneous breathing can be easily initiated by the intravenous injection of 5 ml of nikethamide (Wyant, 1953), and once so initiated will continue spontaneously; also not infrequently it is possible to re-establish spontaneous respiration by the simple expedient of applying oral suction to the endotracheal tube thus over-deflating the lung. Surely neither of these two methods would be of avail in restarting and maintaining spontaneous respiration were apnoea due to an overdose of anaesthesia or relaxant. As a matter of fact, these manoeuvres may well serve as a means of establishing the true cause of prolonged apnoea.

GORDON M. WYANT,
Saskatoon, Canada

REFERENCE

Wyant, G. (1953). "An Indication for Nikethamide," *Anaesthesia*, **8**, 56.

THE ACTION OF LAUDEXIUM

Sir,—Apropos Dr. Hunter's remarks about my letter, published in your June number, my purpose in writing was to evoke accuracy not sympathy.

RICHARD BODMAN

SOCIETY NEWS

THE YORKSHIRE SOCIETY OF ANAESTHETISTS

THE Yorkshire Society of Anaesthetists held its July meeting in Leeds on July 27, when Dr. D. Melrose of the Postgraduate Medical School delivered an address on "Hypothermia". He dealt lucidly with the subject and answered questions from both surgeons and anaesthetists.

The Society was very pleased to welcome Messrs. Allison and Wooler, thoracic surgeons, to this meeting.

The next meeting will be held in October when Dr. H. Churchill-Davison will speak on "Abnormal Responses to Muscle Relaxants".

FACULTY NEWS

COURSES

The Faculty will be holding another intensive three-week course in Anaesthetics at the College from November 28 to December 16, 1955. The course will consist of forty lectures, nightly discussion groups under the leadership of eminent anaesthetists, and visits to London hospitals for medical clinical conferences and demonstrations in practical anaesthesia. Fees: Lectures £15 15*s*; Discussion Groups £15 15*s*.; Clinical Conferences £5 5*s*.

A two-week course of evening lectures in Pharmacology will be held from October 31 to November 11, 1955. Fee £6 6*s*.

Application for these courses should be made to Mr. W. F. Davis, Secretary, Faculty of Anaesthetists, Royal College of Surgeons, Lincolns Inn Fields, London, W.C.2.

MEDICAL GAS: A NEW SAFETY MEASURE

ONE of the remaining risks involved in the administration of medical gases will be removed when a new British Standard system of non-interchangeable valves for gas cylinders and anaesthetic apparatus is introduced. The principle adopted is very similar to that used with radio valves, whereby spacing of the pins ensures that each type can be inserted only in the corresponding socket. Applied to gas cylinders, it prevents a wrong cylinder of gas being connected to the administration apparatus.

Several standard precautions to reduce the possibility of error are already practised. Gas cylinders must be clearly labelled with the names of the contents, and a colour code system is also used to distinguish one gas from another. In some types of connections a special screw thread is used to prevent wrong attachments. The new system will still further reduce any possibility of error.

THE NEW VALVE

The new system is fully described in a revised edition of British Standard 1319—Medical Gas Cylinders and Anaesthetic Apparatus. The system employs the principle of differently spaced pairs of holes in the valves which register with corresponding pins in the connecting part on the administration apparatus.

The connection or yoke on the apparatus will have pairs of pins spaced in conformity with the holes in the valves, so that only the right cylinder can be connected to the right yoke. By this means, for example, it will be impossible to connect a cylinder of nitrous oxide to the administration equipment for oxygen.

An equivalent standard is already in operation in the U.S.A. and Canada, and it is also widely used in Australia and Switzerland. It is hoped that it will be adopted by all N.A.T.O. countries, leading to closer co-ordination between the medical services of their armed forces.

At a meeting of the appropriate Technical Committee of the International Organization for Standardization (ISO) held a week or so ago in Stockholm, the system was accepted as a draft ISO recommendation.

From the coming autumn new equipment of the pin-index type will become available in the U.K., and from the same time manufacturers of gases will make available sufficient new cylinders to meet demands from the users of this new equipment.

THE CHANGE-OVER

The quite separate task of converting the large number of existing anaesthetic apparatus and cylinders cannot be started until about the Autumn of 1956. According to the manufacturers affected, this interval will be required to manufacture the wide range of special jigs, tools and conversion kits required; to build up the trained staff and fleet of special equipment vans needed, and to make sure that there will be available an adequate supply of cylinders with the new valves.

The conversion is being carefully planned on a regional basis, so that apparatus in complete areas are changed over progressively. This will simplify cylinder allocation and distribution. Because of the size of the project, users should take advantage of the change-over service while it is in their area, as otherwise there may be delay before the service van could go to the same region again.

It is proposed that priority of conversion be given to hospitals, followed by equipment used by doctors, midwives, dentists and other professions. The plan will be kept as elastic as possible, and will be adjusted in the light of experience.

It will be seen that conversion of apparatus will have to be carefully co-ordinated with cylinder conversion in order to ensure maintenance of the gas supply service.

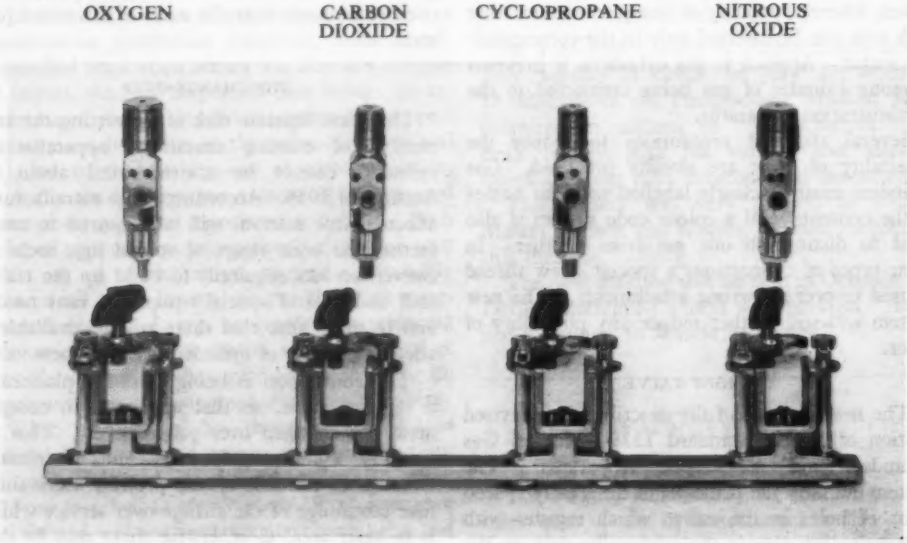
WHO PAYS?

The cost of converting cylinders will be borne by the manufacturers of the gases, but the cost of converting apparatus to fit the new type cylinder

valves will be the responsibility of the owners of the equipment. In some cases the equipment itself may be so old that complete replacement would be advisable.

The new system applies only to cylinders having a capacity of 24 cubic feet or less. Large cylinders are already equipped with valves which make it impossible for the wrong connection to be made. Since pipeline supply systems, such as are used in many hospitals, are fed from the larger cylinders, no question of changing valves or fittings arises.

British Standard 1319—Medical Gas Cylinders and Anaesthetic Apparatus, is published by the British Standards Institution, 2, Park Street, W.1, and can be obtained for 5*s*. post free.



NEW NON-INTERCHANGEABLE VALVE SYSTEM FOR MEDICAL GAS ADMINISTRATION

An assembly of four valves and matching yokes as they will be when the new pin-index system of non-interchangeable valves is introduced into this country. Two pins on the yoke and corresponding holes in the valve—in different positions for different gases—will make it impossible to connect accidentally a cylinder of gas to the wrong place in the administering apparatus.

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